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DENTOFACIAL RELATIONS AT TWELVE YEARS*

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THE strides taken in preventive work in the last two decades should not be regarded merely as a reform in educational principles but as an end product of a widespread realization of the importance of preventive measures in the conservation of human resources. The logical time to begin preventive measures is with youth, for youth is more important than manhood or womanhood, and childhood is more precious than either. If we would only respect the intentions of nature during periods of growth and development, the problem of prevention and correction would not be so serious. In many methods employed by the health service professions a standard, an average, or a norm is essential as a basis for a diagnosis. We already possess norm charts for children at certain ages for height, weight, strength, grip, etc. Every part of the body has a certain order of development with its own critical periods. Periods of growth at which norm patterns of dentofacial relations have been made are at four, six, and eighteen years, and one at a variable age.† If the child is judged by size alone, it might be looked upon as being similar to the adult, only smaller. This is very far from the truth, for the child differs from the adult in every fiber, bone cell, blood cell, and in the relative proportion of all parts.

In this investigation an attempt has been made to arrive at a norm pattern for the denture at twelve years. Twelve years is a critical growth period inasmuch as all the deciduous teeth have been shed, the second permanent molars have usually erupted and have an important influence on the denture. Two

*This investigation was conducted at Washington University School of Dentistry with the aid of a grant for dental research from the Carnegie Corporation.

†These norms were made respectively by Boxton-Olson, Mable Martin, B. E. Lischer, and Paul W. Simon.

hundred and seventy-five children of the St. Louis County schools were examined by direct observation. Thirty-two were selected for the investigation (Table I). All selected had normal anterior-posterior relation of the buccal cusps of premolars and molars when the jaws were in centric occlusion and all permanent teeth had erupted except third molars. The children were selected from homes of ordinary means but had favorable living conditions. Unfavorable living conditions cause a delay in puberty and the abbreviation of the period of accelerated adolescent growth.

TABLE I*

HISTORY OF CHILDREN AND FAMILIES

SUBJECT	NO.	AVER.
Age of mother at birth of child	–	26-27
Age of father at birth of child	–	31
Number of girls in group	18	–
Number of boys in group	14	–
Natal position of child	–	2-3
Number of children in family	–	4-5
Rheumatism in family	11	–
Nervousness in family	8	–
Heart disease in family	3	–
Tuberculosis in family	–	–
Other diseases	–	–
Parents in good health	53	–
Parents in poor health	11	–
Parents with good teeth	27	–
Parents with poor teeth	37	–
Measles in childhood (children)	28	–
Chicken pox	19	–
Scarlet fever	7	–
Diphtheria	4	–
Tooth brush—once a day	16	–
Tooth brush—more	11	–
Tooth brush—seldom	5	–
Visits to dentist—once	10	–
Visits to dentist—more	14	–
Visits to dentist—never	17	–
Bottle-fed babies	17	53%
Breast-fed babies	15	47%

*All information in this table was based on the testimony of the parents. The descent of these children was from German, English, Irish, Scotch, Swedish, and French stock, although the German predominated. All history of the deciduous teeth was favorable.

METHODS OF INVESTIGATION

An oral prophylaxis was given each child, and a carefully made dental record was charted. The orbitalia and left gonion were marked.† Photographs, one-fourth size, were taken with a National photognathostatic camera. The anteroposterior diameter of the maxillary incisors was recorded to be compared later with Pont's index of arch width. Gnathostatic impressions were taken, and gnathostatic reproductions were made (Fig. 1). Three-dimensional graphs were then drawn for each of the thirty-two reproductions (Fig. 2), and photographic left profiles were marked in accordance with Simon's recommendations. Complete dental radiographic examinations were also made, which included sixteen intraoral views.

†When the patient is looking directly forward, the orbitalia are located on the highest point on the orbital ridge directly under the pupils of the eye. The gonion is located at the angle of the jaw.

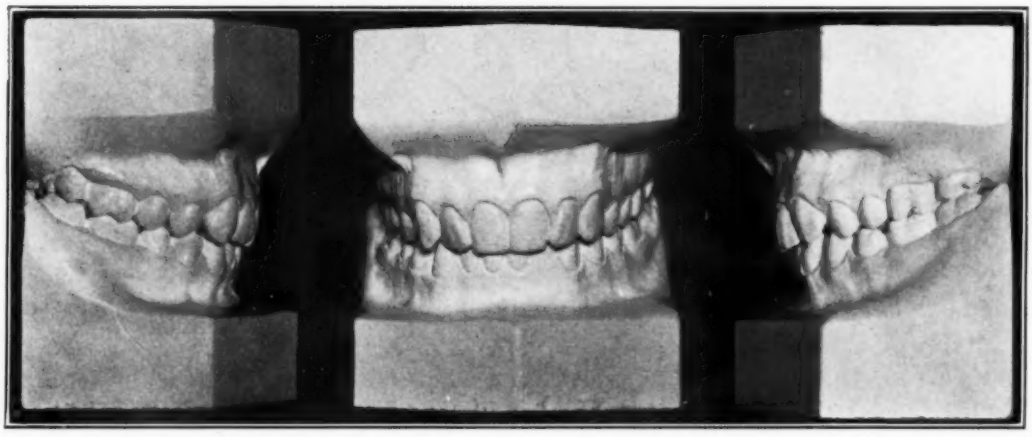

Fig. 1.—Gnathostatic reproductions.

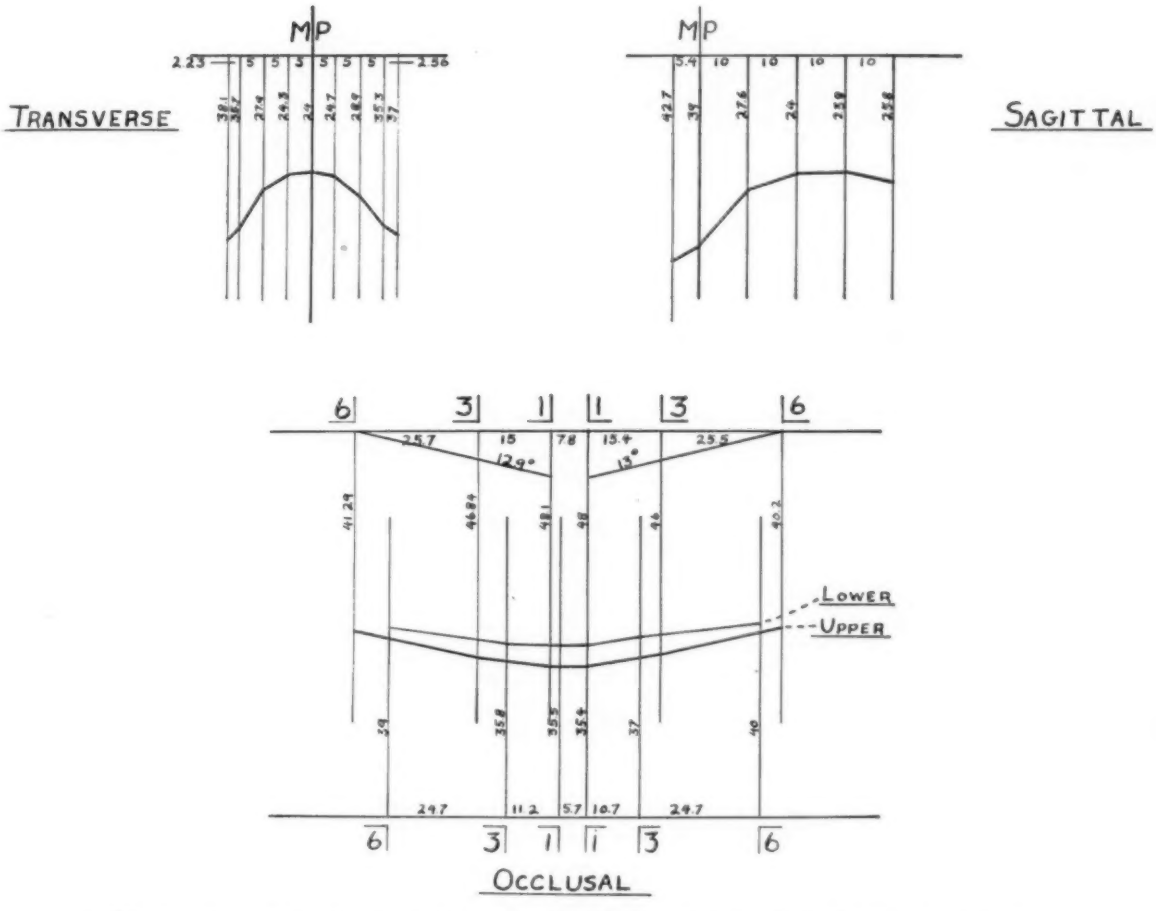

Fig. 2.—Three-dimensional graph of 12 year pattern. Norm pattern 12 years 7 months.

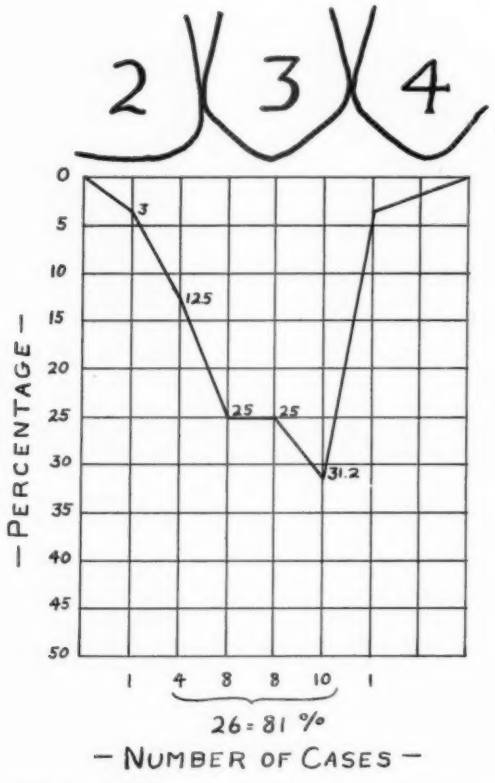

Fig. 3.—Position of orbital plane in relation to the canine. Sagittal relation diagram.

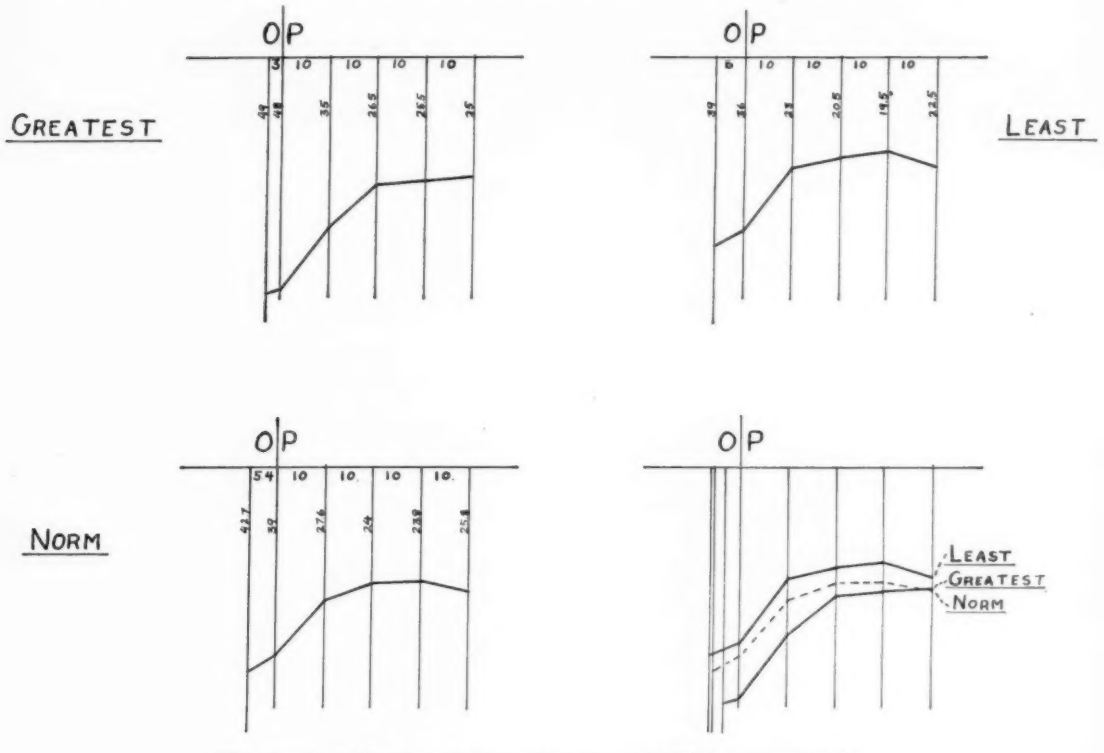

Fig. 4.—Length of prosthion ordinate. Sagittal curve, height.

FINDINGS OF THIRTY-TWO SELECTED CASES

I. *Gnathostatic:*

A. Median Relation. (a) The denture was symmetrical in regard to the median raphe in fourteen cases (43 per cent); twelve cases (37 per cent) presented deviations from 1 to 2 mm. and were called "physiologically symmetrical." Four dentures (10 per cent) revealed marked asymmetry.

B. Transverse Palatal Curve. (a) The greatest width of the transverse palatal curve, which is customarily located 2 cm. posterior to the orbital plane, was 38 mm.; the narrowest was 30 mm.; the average width of the series was 34.79 mm. (b) The greatest median height of the palatal curve was 17 mm.; the lowest was 9 mm.; and the average was 13.5 mm.

C. Relation of the Denture to the Orbital Plane. (a) In twenty-six cases (81 per cent) the orbital plane passed through the canine, and in six cases (19 per cent) the orbital plane missed the canine (Fig. 3). (b) The greatest degree of prognathism, measured from prosthion to orbital plane, was 13 mm. The average for the series was 5.4 mm.

Fig. 5.—Position of the cheilion to orbital plane.

D. Relation of Denture to the Eye-ear Horizontal Plane. (a) The greatest median height of the upper occlusal curve was 12.5 mm.; the least was 2.5 mm.; the average for the series was 7.5 mm. (b) The greatest amount of inclination of the maxillary occlusal curve was 23 degrees, the least was 4 degrees, the average for the right side was 12.9 degrees and left was 13 degrees. (c) The length of the ordinates from the horizontal plane: the greatest prosthion ordinate was 49 mm., the least was 39 mm., and the average for the series was 42.7 mm. (Fig. 4). The greatest orbital plane ordinate was 49.5 mm., the least was 35 mm., and the average was 39 mm. The greatest ordinate length taken 2 cm. posterior to the orbital plane was 29.5 mm., the least was 21 mm., and the average was 24 mm. The greatest ordinate length at the posterior of the denture was 32.5 mm., the least was 20.5 mm., and the average for the series was 28.5 mm.

II. *Photostatic:*

A. Profile Contours. (a) The position of the cheilion in twenty-seven cases was anterior to the orbital plane; it fell slightly posterior to the orbital plane

in three cases, and fell on the plane in two cases (Fig. 5). (b) The gnathion position in eighteen cases fell on the orbital plane, twelve cases were slightly posterior, and in two cases the gnathion fell markedly posterior to the orbital plane (Fig. 6). (c) There was a parallelism presented in twenty-two cases between the oral fissure and the horizontal plane, nine cases showed a divergence posteriorly from the horizontal plane. Variations were found in the upper lip curve, in the length of the upper lip, and in the lower lip curve.

B. Profile Measurements. (a) The greatest tragion angle was 80 degrees, the least was 63 degrees, and the average was 72.7 degrees (Fig. 7). (b) The greatest gonion angle was 156 degrees, the least was 124 degrees and the average for the series was 133.6 degrees (Fig. 8). (c) The greatest length from the orbitalia to the tragion was 21.5 mm., the least was 19 mm., and the average was 20.3 mm. (d) The greatest length from tragion to gonion was 16.5 mm., the least was 9.5 mm., and the average was 13.9 mm. (e) The greatest length from gonion to gnathion was 20 mm., the least was 10.5 mm., and the average was 16.2 mm. (f) The greatest facial height, measured from nasion to gnathion, was 24 mm. and the least was 19 mm.

III. *Radiodontic:*

A. Bone: In classifying bone, we divided it into dense, normal, and thin. By dense bone we mean that bone that has a large number of trabeculae which are finely spaced. Normal bone is bone that has a uniform and even arrangement of trabeculae and average density. Thin bone is bone that has very few trabeculae and many large medullary spaces. In examining the radiographs we found no dense bone, which seems to be a logical finding for children of this age. Twenty-three children demonstrated normal bone (72 per cent), nine children showed thin bone (28 per cent).

B. Caries and Restorations: The dental caries appearing in the radiographs was divided into active and recurrent. Seven active interproximal cavities, eight active occlusal cavities, and four showing recurrent caries were found. Out of the 996 teeth present in these children, 1 per cent showed dental caries of less than one cavity per person. Out of the 996 teeth present, 2 per cent had restorations or a little better than one restoration per person. Pulp involvement which needed treatment was found in two cases and root canal fillings were found also in two other cases. The position of the third molar appeared unfavorable for eruption in only one case, that is, it already appeared to be impacted. This tooth was a mandibular left third molar.

CONCLUSIONS

Good occlusion tends to increase ability to masticate food properly. Proper mastication not only prepares food for swallowing but trebles the amount of saliva which is mixed with food. Proper occlusion aids in mastication and stimulates healthy development of the jaws and growth of the teeth. Good, well occluded teeth tend to clean themselves, and finally, caries seems to be decreased as the small amount of less than one cavity per person indicates. The average school child has from three to five decaying teeth. Norm patterns

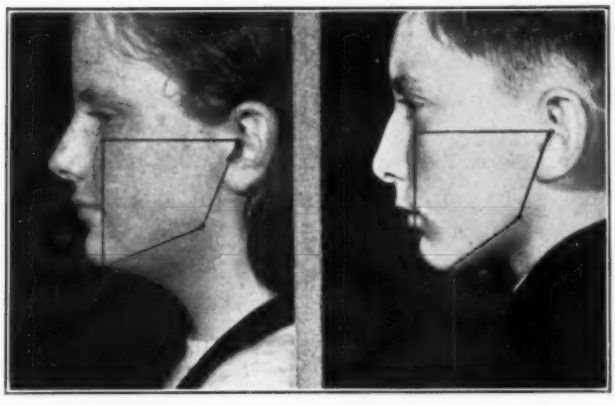

Fig. 6.—Variation of gnathion position to orbital plane.

Fig. 7.—Variation of tragion angle.

Fig. 8.—Variation of gonion angle.

aid in diagnosis, and the only way to truly possess knowledge and benefits from them is to use them as one of the several helpful aids in a complex problem.

The author wishes to express his appreciation to Drs. B. E. Lischer, W. E. Koch, Jr., and J. A. Laffler for their invaluable assistance.

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Medical Arts Building

NUTRITIONAL CORRECTIONS AS AN AID IN OVERCOMING GROWTH DEFECTS IN THE ORAL STRUCTURES*

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HIPPOCRATES, the Father of Medicine, living from 460 to 377 B.C., is reported to have stated: "There are many other ills different from those of repletion, but no less dreadful, arising from deficiency of diet."

Weinberger[1] in a recent article on "Medical Problems Relating to Orthodontia" reports that Hippocrates also observed "among those individuals whose heads are long shaped, some have thick necks, strong members, and bones; others have strongly arched palates, thus teeth are disposed irregularly, crowding one on the other. . . ." He further states that there is sufficient evidence to show that Neanderthal Man, of 40,000 to 60,000 years ago, suffered from irregularities of the teeth, supernumerary, retained, and impacted teeth much as man does today. Hence we see that neither nutrition problems nor orthodontic problems are of recent date.

Before discussing my subject, may I present a few other points of view which are fundamental to the concept of orthodontics, and which in turn may or may not upset an excellent nutrition program?

Dr. Weinberger pleads for broader training for students of orthodontics. He cites the following subjects as those which should be considered fundamental to this science: "anthropology, general and physical; biometrics; growth and development; education; engineering; endocrinology; genetics; habits and psychology; heredity and inheritance; metallurgy; nutrition; pediatrics; structural deformities; orthopedics and surgery." He states: "No one can say after such a grouping that orthodontia is just a problem of moving teeth. It is the connecting link between dentistry and medicine, extending into anthropology, physics, chemistry and art."

What does each of the sciences listed by Dr. Weinberger have to contribute to orthodontics? Time does not permit discussion of the role of each, but the importance of a few will be mentioned.

Professor E. A. Hooton[2] in an article entitled "An Anthropologist Looks at Medicine" states: "Anthropology reveals many things which most persons prefer not to know, since it harps upon humble and even bestial origins, regards the present status of our species without approbation, and can predict for the man of the future no apotheosis, but only a multiplication of psychoses, dental caries, malocclusions and fallen arches, together with a full retention of his aboriginal cussedness. . . . Not for the anthropologist is the contemplation of the celestial grandeur of the expanding universe; he looks at one animal 'bereft of

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his tail and grown weary of climbing'; he wonders why that animal carries so much excess baggage in the way of apparently unutilized brain, and whether he would not be better off with a smaller hat size and a set of lower and upper teeth which hit.'' According to definition in Dorland's ''American Illustrated Medical Dictionary'' (17th Ed.), ''dental orthopedics'' is defined: ''correction of deformities or irregularities of the teeth; orthodontia of children's teeth.'' Hence, if orthodontics is in reality a branch of orthopedics these statements of Dr. Hooton are apropos.

C. M. Waldo[3] in his paper ''Orthodontic Research as a Component Part of a Balanced Longitudinal Study of 100 Children'' stresses the need for research on growth and development. He comments as follows: ''Our major problems differ only superficially from those of other branches of medicine. The unanswered questions which puzzle us are in many instances the same ones which puzzle the pediatrician or the otolaryngologist, and most of them have a common source—our lack of knowledge of human beings, more specifically the growing child. Our situation is similar to that of a person trying to translate something written in an unfamiliar language of which he knows only a few words. No amount of effort is likely to give him the meaning of what is written until he enlarges his vocabulary. The information which we need is as fundamental and as indispensable as an adequate vocabulary, and it is the task of research to supply this working knowledge which will place us in a position to attack our new problems with success. The longitudinal method of study of individual children seems to offer the greatest opportunity for building up this picture where variation in structure or function ceases to be a normal or healthy deviation and becomes pathologic. A knowledge of the spread of the normal range in the growth and development of children is as essential to the orthodontist as to the pediatrician.''

A most remarkable book and one of interest to every orthodontist was called to my attention by Dr. Guy S. Millberry. It is recommended to Dr. Millberry by Dr. J. B. S. Haldane, eminent geneticist of the University of London, as being the outstanding book on inheritance of dental abnormalities. It was compiled by Dr. E. A. Cockayne of London and is entitled ''Inherited Abnormalities of the Skin and Its Appendages.''[4] A few of the chapter headings are indicative of the scope of the book. One entire chapter of 14 pages is devoted to Abnormalities of the Teeth as they occur in families, including absence of various teeth, absent or deficient roots to the teeth, hypoplasia of the enamel, early dental decay, microdontia, and supernumerary teeth. Other chapter headings are: Dental Abnormalities Associated With Major Disorders of Development; Metabolic Errors; Ectodermal Dystrophies; Abnormalities of the Nails; and Disorders of Growth. It may be said in passing that the more one studies this book, the more amazing it appears that any of us arrived in this world with most parts of our anatomy in the right places.

M. F. Guyer[5] in his paper ''Heredity and the Constitution of the Child'' says: ''This brings up the whole question of internal secretions as factors in development. Height, broad or slender form, length of arms and legs, shape of face, quality of voice, distribution of hair or of fat on the body, and even emotions are in greater or less measure conditioned by the relative functioning

of the various endocrine glands during the earlier development and later life. And there is no reason to doubt that the amount and quality of the secretions in various family strains are as much the expression of hereditary factors as are many other individual characteristics. The hereditary aspect of these glands, however, is likely to be overlooked because they are also subject to environmental modifications and because one is accustomed to think of them in terms of their immediate activities instead of their genetic constitutions."

The following quotation from the excellent paper on the "Integral Growth of the Face" by Todd and Broadbent[6] is pertinent. They state: "Everyone knows, of course, that faces differ and most of us are content to assume that the differences are largely due to the hereditary tendencies in the genes. But the face like the rest of the body is a plastic thing and since the adult contours are the end result of a growth pattern which in the course of its progress may be expedited, interrupted, retarded, warped, or inhibited by misadventures of health and vagaries in the interplay of those organically originated influences by which the pattern is promoted, it is evident that environment, external and more particularly internal, must contribute in no small manner to the final result. This has been emphasized in recent years by Howard[7] and the growth pattern itself propounded in detail by Hellman[8]." Time permits only mention of the contribution to orthodontics by the interesting study of Rosenberger on "Growth and Development of the Naso-respiratory Area in Childhood" made possible through the courtesy of Dr. B. Holly Broadbent, director of the Bolton Study Anatomical Laboratory, Western Reserve University.[9]

In 1923 Hellman[10] speaking before another group of orthodontists began his paper on "The Process of Dentition and Its Effect on Occlusion" with the following quotation from the book by Dr. L. L. Woodruff entitled "Foundations of Biology": "In ultimate analysis everything is incomprehensible, and the whole object of science is simply to reduce the fundamental incomprehensibilities to the smallest possible number." Even this sketchy résumé of a few papers makes it apparent that much progress has been made in the understanding of the etiological factors concerned with orthodontic problems since Hellman quoted this statement fifteen years ago.

Perhaps you are wondering why time has been taken from a paper entitled "Nutritional Corrections as an Aid in Overcoming Growth Defects in the Oral Structures" to present this apparently irrelevant material. As a result of researches in nutrition during the past twenty-five years there is a great deal of interest in this subject. Paradoxically this in turn has brought about a lack of understanding on the part of many individuals of what optimum nutrition can and cannot accomplish. If one were asked to discuss the role of nutrition in beef, wool, mutton, egg, or milk production there would be no justification for the time spent for the reason that in animal industry desirable attributes such as ability to produce large quantities of high quality meat, milk, wool, butter or eggs *must be* in great measure *bred* into the animals, only to a limited degree can they be *fed* into them; in other words, heredity is first and nutrition second in importance. No animal breeder would expect a scrub bull to develop into a prize animal even though fed a highly satisfactory diet, or a low milk producing

cow to develop into a high producing one by being given an optimum diet. Nevertheless, the diet of pedigreed bulls, stallions, dairy cattle, beef cattle, and other high grade stock is planned carefully because optimum nutrition unquestionably aids in developing the hereditary potentials of these animals. It would, indeed, be poor business to feed valuable livestock in the haphazard way that is adopted in the feeding of many children and also many adults.

Of what value then is this discussion to the orthodontist? Unlike the stock breeder who is able to discard all animals which do not suit his purpose, the orthodontist must work not only with individuals who are the best possible risks but also with those persons with mixed inheritances and with various health and nutrition histories. Papers under the titles "Nutrition—One Factor in Orthodonties" and "The Nutrition History and Its Relation to Dental Problems" I have already published.[11]

What is known in nutrition as the Law of the Minimum advanced by the German chemist, Justus von Liebig, in 1843 is a most fitting basis for an approach to a discussion of nutrition as a factor in orthodontic problems. This law is generally taken to mean that "among the substances nutritionally essential for growth that one which is furnished to the organism in minimum amount (relative to the need for growth at the normal rate) will thereby determine the rate of growth, the organism growing only to the extent that it can increase in size and at the same time conform to the normal composition of its kind." The truth of this law from the protein standpoint was strikingly demonstrated years ago by Osborne and Mendel[12] of Yale University. They fed little rats diets satisfactory in all respects except that the protein supplied was low in the amino acid, lysine. Those investigators found that the animals grew only in proportion to the amount of lysine which the food supplied. Instead of growing up with lysine-poor bodies, the animals grew at the rate at which their bodies could make new body proteins of normal lysine content and then stopped growing. For example, little rats after being on the lysine-poor diet for 276 days weighed only about 70 grams, whereas rats on an optimum diet for 276 days weighed about 325 grams or more.

The Law of the Minimum is applicable *to proteins* as just shown, but that it is not applicable *to minerals* was demonstrated by Sherman and his coworkers at Columbia University. They fed four groups of rats diets that varied only as to the calcium content. They found that the increase in body weight was practically the same for all groups of rats but the rate of calcification of the skeletons was markedly different; this was demonstrated by ashing the skeletons.[13] In other words, while the body will not grow to full adult size with lysine-poor proteins, it may grow up with calcium-poor bones and iron-poor blood. Sherman so aptly states: "The iron-poor body is almost certain to show anemia; this commands attention and sympathy; doctors, nurses, parents, and teachers recognize an anemic condition and proceed to remedy it. But under the same sympathetic observers, a correspondingly calcium-poor condition of the body may go unrecognized for an indefinite length of time."[14]

As evidenced by roentgenograms, many children are growing up with poor bone structure, which in turn undoubtedly makes them poorer orthodontic risks than they would otherwise be.

The following excerpts from a paper by T. Wingate Todd[15] on "The Mineralization Problem in Orthodontics" states the problem at hand excellently. Todd says "The mineralization problem of the orthodontist implies much more than the provision of an adequate mineral ration for the child. Stated baldly it means that the orthodontist must rely upon the minerals of the jaws securely fixing in their new alignment the teeth which he has rearranged. The teeth are not sunk in posts of cement. They are more correctly likened to a team of horses whose efficient cooperation depends upon the bridle of occlusion. The harness must be strong enough to hold the teeth in position but it must also be resilient enough to ease off here and there as the strain of the work requires. One must realize that in mineralization one is not studying the construction parts of a permanent substance but rather the fabrication of a supporting framework, the pattern of which may indeed be permanent but the constituent parts of which are constantly undergoing change and reconstruction to meet the needs of the moment in alignment and activity. Our problem then divides itself into estimation of two factors: namely adequacy of structure and adequacy of response. . . .

"Several minerals enter into construction of bone but since bone contains about 99 per cent of the calcium in the body, calcium is considered the chief of minerals. . . . Children of impoverished constitutions, whether from prolonged toxemia, protracted ill-health or inability to utilize minerals, show pronounced reduction of the labile minerals with encroachments even on the trabeculae themselves which become thinner or fragmented. . . . Demineralized bones of this type are not defective in their potentiality for repair. They fracture easily but they form callus though of a demineralized type. Bones such as these are simply inadequate not perverted in functional character. . . . It is the demineralized bone which so impedes the work of the orthodontist. There is no efficient response which the orthodontist can call to his aid in fixing the realignment of the teeth brought about by the expenditure of the orthodontist's time and the patient's patience."

In addition to (1) adequacy of structure and (2) adequacy of response emphasized by Todd, I would stress (3) adequacy of building materials; i.e., abundance of all dietary essentials needed for optimum health, but calcium and phosphorus and vitamins C and D should be given special emphasis by the orthodontist.

The work of Wolbach and Howe[16] on vitamin C is of interest to all dentists, including orthodontists. These investigators have shown that "in vitamin C deficiency there is an inability of the supporting tissues to produce and maintain intercellular substances. . . . The intercellular substances concerned in vitamin C deficiency are collagen of all fibrous tissue structures, the matrices of bone, dentine and cartilage, and all nonepithelial cement substances, including that of all the vascular endothelium. . . . The important gross features of scurvy at all ages are hemorrhages and changes in the bones. The most striking lesions are the subperiosteal hemorrhages. . . . Hemorrhages are due to mechanical weakness, occasioned by the lack of collagenous material in fibrous tissue structures and in bone. Diminished cohesion of endothelial cells contributes to the ease with which moderate stresses occasion bleeding in skin and muscles."

Even though an orthodontist would not be working on a child having scurvy, the data of Wolbach and Howe are of interest, because it is now recognized that many persons, including children, take a diet too low in vitamin C for optimum well-being.

The importance of vitamin D to calcium and phosphorus metabolism is too well known to need discussion here. If the diet suggestions published elsewhere[11] are followed, not only will an abundance of calcium and phosphorus but also other dietary essentials be present in the diet. That children may eat an adequate diet and yet be below par due to inability to assimilate various dietary factors is well known. These children should of course be under the care of a physician.

As a result of our studies at the College of Dentistry at the University of California, what can be said of the role of nutrition as an aid in overcoming growth defects in the oral structure?

Before making a few remarks I want to thank Dr. Guy S. Millberry, our Dean, whose keen interest in nutrition and its relation to dental problems made possible this cooperative study. I also want to thank Dr. Ernest M. Setzer, Chairman of the Department of Orthodontics and the men and women on his staff for the interest they have shown and the "private education" on orthodontic problems which they have given me; Dr. H. Becks of the Department of Dental Medicine and Dr. S. T. Pope, Jr., the physician in our group, for their helpful suggestions in their respective fields. The time has not come for a complete report on these studies but a few comments are warranted.

Dr. Setzer and I checked the present status of 58 patients who have been under observation between three and four years. They have had all active appliances off for varying lengths of time. Before commenting upon our findings it should be emphasized that any study of this kind cannot be viewed with the coldly critical eye turned to view animal research for the reason that no controls are available. No animal work is ever done or should ever be done without an abundance of controls.

Even if an orthodontist were fortunate enough to have 50 sets of identical twins as patients with the same degrees of malocclusions and each set of twins had had the same number, kinds, and degrees of illnesses; had eaten approximately the same kind and amounts of food; and of course would have the same orthodontist during the course of treatment, each child would not be a control for the other in the true sense of the word, because of variables mentioned in the early part of the paper. In nutrition work with animals selective breeding and ruling out of unfits not only make controls possible but also enable one to *prove* that a certain dietary factor did or did not improve the animals because the one factor in the diet is the only variable. A moment's thought shows how impossible such controlled nutrition experiments are with persons.

In our studies we have utilized student work in the college clinic, under supervision of course. Due to transfers, graduations, etc., in most instances several students have worked on each patient. As every one knows, there are variations in the abilities of students. We were also working with a group of children from the income level group where a highly satisfactory diet was the exception rather than the rule. Dietary recommendations were made, but in

many instances only Fair to Good cooperation was given due in part to the added expense. We have had, therefore, several variables in our work which would not be present in a similar study made in private practice.

Dr. Setzer rated the present status of the cases under the following headings: (1) condition of the soft tissues; (2) occlusion; (3) nature of the result.

A point of great interest is that several of the children I had rated in my early notes as Poor or Fair risks because of their health and nutrition histories were rated at the time of the check-up by Dr. Setzer as Good to Excellent under the above headings. A check of the students who had worked on these cases almost invariably showed that unusually capable students had been working on these children. Several of the children whom I had rated Good or Excellent risks from their health and nutrition histories were found by Dr. Setzer to rate only Poor or Fair under the above headings. Again a check of the students working on the cases almost invariably showed that less capable students had for the most part been working on these cases. In numerous instances, however, patients with good to excellent health and nutrition histories rated good to excellent on the above ratings. Fortunately from the research standpoint except in a few instances the patients who were poor risks from the nutrition standpoints were not worked upon by the less capable students and vice versa. No selection of patients, however, was consciously made.

A few sentences from the splendid paper by de Vries[17] on "What Are the Possibilities and Limitations of Orthodontic Treatment?" would appear to be in order at this point. He states: "The possibilities of orthodontic treatment seem limited to the same degree as the particular human organism is limited in its intrinsic ability to attain the maximum of developmental and functional perfection. . . . Not all persons possess the same capacity for attaining a standardized normal."

A most interesting part of our study will be a further check-up in a year or so to learn whether those children rated originally as Poor or Fair risks from their health and nutrition histories will remain in the good condition, orthodontically speaking, that they are now, especially if they slip back into their earlier unsatisfactory food habits. They have been counseled, but as stated before Fair cooperation due in part to added expense is about all that can be expected. In private practice better cooperation could probably be secured in part because you are working with a higher income group. A more detailed report of our studies will be given later.

Nutrition has been said to be "an agricultural, economic, commercial, and industrial problem as well as a problem in physiology."[18] I would like to add that it is a problem in education, which in the last analysis is probably the most difficult one to handle.

In closing then, what has nutrition to offer in overcoming growth defects in the oral structure?

Although our study is far from completed, it has shown thus far that nutrition must be viewed with perspective in orthodontics just as it must be viewed with perspective in other fields such as preventive medicine and public health. It is only one factor in a large problem.

Optimum nutrition before or during orthodontic treatment will not take the place of the orthodontist's skill and judgment; there are no substitutes for these qualifications.

But if all patients have had in the past and have during and after treatment an optimum diet and a healthy body which enables them to utilize the various factors in their diet, the orthodontist would have one less variable with which to contend. Malnutrition with or without rapid growth cannot be other than a poor combination for an orthodontist. Is not Todd correct when he says that "It is the demineralized bone which impedes the work of the orthodontist"? This type of bone may be and often is the result of a faulty diet, but of course factors other than diet may bring about a demineralized bone.

Our studies have shown further that in order to give a child all the assistance possible during and after orthodontic treatment, at least a brief health and nutrition history should be taken on all patients. If necessary their diet should be adjusted so that all factors are abundantly supplied. Time, cost, convenience, habits, likes, and dislikes are a few of the factors which must be taken into consideration when dietary suggestions are made. The essentials of an adequate diet and one procedure for taking a dietary history have been discussed.[11] A discussion of other dental problems and medical problems associated with orthodontic problems is outside my field.

Due in part to the fact that research in nutrition has not only demonstrated the existence of vitamins but also isolated and synthesized several of them, much of the world is extremely vitamin conscious at the present time. The various vitamin preparations (capsules and tablets), dicalcium phosphate wafers, and other calcium salts have been given a great deal of publicity, which has tended to focus too much attention upon these products. It should be emphasized that these products are not panaceas which will solve all nutrition problems, valuable as they are for supplementing the diet when certain foods are disliked. But persons should know what they *do not* contain as well as what they *do* contain. As I have stated elsewhere the garden, the grocery, the orchard, the dairy, the butcher shop, and the great out-of-doors should be depended upon in great measure to supply our dietary needs rather than the drugstore.[19]

There may be times when it may be advisable to resort to dicalcium phosphate wafers or capsules or other calcium salts to increase the calcium and/or phosphorus content of a child's diet, but consultation with a physician is recommended before doing so for the reason that if a child is unable to take milk and its products, eggs and/or meat in sufficient amounts to supply most of the calcium and phosphorus needed he (or she) should be under the care of a physician. Such a child is a medical problem in addition to an orthodontic problem.

To summarize: It may be said that a highly satisfactory diet is one of the building stones for optimum health, but many factors other than diet play a role in the health set-up.

All children are not equal orthodontic risks from the health and nutrition standpoints.

All other things being equal, a person in optimum health should be a better orthodontic risk than a person in poor health.

But an optimum diet and abundant health can in no way replace a well-trained and a well-informed orthodontist, who possesses not only technical skill but also good judgment; they only supplement him.

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WHEN IS THE FRENUM LABIUM A PROBLEM IN ORTHODONTICS?

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THIS brief paper is really an expression of my own opinion based upon many years of observation and clinical experience and a reasonable familiarity with the literature on the subject. Right or wrong, I do not hesitate to answer the question implied in the title by saying that the superior frenum labium is a problem in orthodontics only when it is made so by the operator himself, except possibly in the case of young men who have had little or no experience with it and are also unfamiliar with the literature on the subject, which seems to me already to be adequate. If it is adequate, I suppose I should not be writing this paper; especially as in the past I have gone so far as to say that the subject did not warrant further space in programs and dental journals. In my own defense I may say that I did not suggest this subject for a paper but am only responding to a request coupled with the statement, as a reason for the request, that orthodontists in many sections of the country are still arguing vociferously about it, and indeed I must admit that wherever the subject comes up it seems to be rather hotly contested from the standpoint of widely divergent views.

Actually, the frenum has been a subject of interest, sometimes controversy, for at least forty years. It may have been given some attention in the literature previous to that time, but Dr. Angle brought it conspicuously to the attention of the profession in his writings forty or more years ago. I believe no orthodontist familiar with the development of the specialty since the Angle School was established will deny that Angle has never been surpassed for keen and accurate clinical analysis of the problems with which orthodontists are confronted, and I might add that no one has contributed more nor perhaps as much to the solving of those problems.

Angle recognized that the frenum presented variations in size and attachment. Close observation showed him, what I believe it will show anyone today, that some frenums are abnormal either in their size or in their attachment or both. With characteristic thoroughness he analyzed the effect of an abnormal frenum upon the associated structures; and recognizing the unusual activity of the muscles of the lips during the various functions performed by the mouth, he concluded that a frenum which was attached too low and too deeply could, and actually did, cause a separation of the anterior teeth. Upon recognizing the nature of the abnormal frenum and its effect upon the anterior portion of the maxillary dental arch, Angle[1] immediately devised a treatment which he described as follows in his seventh edition of *Malocclusion of the Teeth:* "With a suitable lancet or bistoury a deep incision is made between the teeth, splitting the ligament, after which an electrocautery knife, . . . at white heat, is

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passed through the incision. No pain will be occasioned if, preliminary to the operation, the tissue be locally anesthetized with a proper solution of cocaine, applied by means of a pledget of cotton for about ten minutes. Great care should be exercised in the use of the cautery instrument, which should come in contact only with the abnormal tissue, the wounding of the peridental membrane being rigidly avoided, and for this reason the clean incision is first made to simplify the operation. Although the contraction due to the cicatricial tissue is considerable, yet when we remember the character and structure of the peridental membrane and the immense number of normal fibers acting to combat this tendency, and that they are practically double in these cases, for two teeth are involved, the necessity of support for a considerable length of time (a year and a half in some cases) should occasion no surprise."

Undoubtedly a great many patients were operated upon in this manner, and there is no doubt but the superfluous part of an abnormal frenum can be removed with an electric cautery. There were, however, some cases reported in which the cauterizing had been too deep, and injury to the periosteum caused sloughing and exfoliation of small sequestra of alveolar bone. A few such reports seemed to create in the minds of many orthodontists the impression that operating on the frenum was quite a serious undertaking and one which was likely to lead to very unfortunate results and therefore one to be avoided. Men began to take the stand that it was an unnecessary operation anyhow and that if the teeth were drawn together and held long enough, the frenum would disappear through pressure atrophy exerted by the roots of the teeth. This does not seem rational to me, and I am very doubtful if the white fibrous tissue contained in abnormal frenums is as likely to absorb as is the root of the tooth. Be that as it may and granting that it would be successful in cases where a long orthodontic treatment is unavoidable anyhow, it certainly could not be justified in a case which required only the correction of the frenum without other malocclusion present, and there are many such cases.

This attitude which pervades a large part of the profession has often prevented patients from getting the benefit of a simple operation which can be performed with less shock and trauma than would be occasioned by the removal of a deciduous molar. I believe it has arisen partly from the fact that our calling is a sort of bloodless surgery, and some are inclined to carry the idea to extremes. This occurs in other specialties. I remember a nose and throat specialist who enjoyed a large practice but who was said never to remove tonsils however serious their condition. One of his confreres said to me, "He is a fine man and a good physician from a treatment standpoint but he simply won't draw blood." Now it is just as reprehensible to operate on tonsils and frenums or anything else that does not need it as it is to neglect to do so in cases that do need it, and both of these faults have been observed in the attitude of the profession towards this very simple phase of orthodontic treatment. I was asked to read a paper on the abnormal frenum problem. Frankly, as I said at the outset, I do not consider it a problem, although I admit a problem has been made of it in the minds of a great many of the profession. It pops up perennially in the literature, and I myself have criticized people for writing articles on a subject which it seemed to me had already been settled several times.

In 1933, George M. Anderson,[2] a former associate of mine, gave a concise illustrated description of the operation for correction of the frenum, and again in 1937[3] he presented the result of a histologic study made by him and M. S. Aisenberg on the frenum and adjacent structures of embryos. This investigation showed that the frenum did not pass through the premaxillary median suture, and while this might be thought to settle the controversy which has existed in the past as to whether the fibers of an abnormal frenum did pass through the suture or not, it does not settle it in my mind except for embryos. I am willing to believe that the fibers under no circumstances ever pass through the suture in the prenatal individual, but I also believe that they do become enveloped by it as the alveolar process grows downward, and in talking with Anderson very recently he told me he thought this quite possible and indeed was inclined to agree with my view. I base my opinion more on the behavior of the structures involved, before and after correction of the frenum, than I do upon radiographic studies which often show ample space for fibers to pass between the lateral halves of the premaxillary bone but cannot register them in a radiographic picture. The deep labial furrow which appears between the alveolar sockets of the two central incisors shows clearly to my mind that not only have the teeth themselves separated but they have reconstructed their socket walls much the same as teeth do adjacent to one that has been extracted. This furrow or groove to a great extent disappears when the frenum has been properly corrected and the anterior teeth have been drawn together. Some writers speak of the scar tissue left by the operation as being a hindrance to perfect maintenance of the teeth in contact. Others, especially those who have advocated the cautery, speak of the scar tissue thus formed as being a great aid in maintaining the teeth in contact. Actually, when the operation is done in a simple but correct surgical manner, it may be said that there is no scar tissue, and certainly it is not visible along the labial portion of the alveolar gum, though it may show slightly on the inside of the lip in some cases. One prominent man (Dr. Meyer L. Rhein) in another specialty, periodontia, advocated the correction of the frenum in pronounced cases for purely prophylactic reasons, saying that the pockets formed on each side of the frenum and high up under the lip were unclean. I have seen cases which I think bore out this contention.

In 1933, Winston P. Caine[4] published several letters written him in answer to a questionnaire he sent out on the advisability of operating on the superior frenum labium. The letters show the usual divergence of opinion on this subject. Dewey's characteristic answer reflects my own attitude precisely. He said, "In reply to your letter I would advise the removal of all abnormal maxillary frenums. I certainly would not remove a frenum unless it was abnormal. I have been unable to get satisfactory results in cases of abnormal frenums without their removal." The idea is to determine whether the frenum is abnormal, and if so, correct it and proceed with orthodontic treatment if it is indicated. One of the most marked diagnostic symptoms of an abnormal frenum is the blanching of the lingual tuft of gum when the upper lip and frenum are pulled firmly away from the teeth. This is recognized by most of the men I have known who believe there is such a thing as an abnormal frenum and that when

it exists it should be corrected. Ketcham in the same questionnaire spoke of "Those rare cases where the abnormal attachment of the frenum is very pronounced or where it is so low that it interferes with proper toothbrush excursions over the labial surfaces of the incisors." It is not conceivable that a child who is tongue-tied, that is, one who has an abnormal frenum under the tongue which prevents it from talking and nursing properly, would be neglected to the extent of not having it corrected. As a boy, however, I did actually know another boy who could hardly make himself understood because of being tongue-tied. This was in the then remote mountains of North Carolina. Why the superior frenum labium should be regarded with such awe and as being so different from the other numerous frenums which characterize the median dividing line between the two lateral halves of the body, I cannot imagine, but such seems to be the case. In the six or seven papers or demonstrations appearing annually in the literature, there are often as many different views expressed, the real issue, however, usually being as to whether the frenum does or does not in some cases cause separation of the anterior teeth. The arguments pro and con remind one of the old controversy over the subject of the artificial opening of the maxillary median suture by means of orthodontic appliances, and are waged quite as hotly.

For many years I operated on such cases as came into my practice and in a few instances for other men. With the advent of the exodontist, or dental oral surgeon, I found it easy to explain to him what I wanted in the operation and also easy for him to grasp it and carry it out, and by referring these cases, I was saved the annoyance of maintaining constantly the rather extensive novocaine paraphernalia required to obtain the best results. It is simple enough if one is using it constantly but annoying and time consuming if one uses it once in a month or possibly once in three months.

The operation, as I performed it, consists in having the assistant hold the lip upward and draw the frenum taut. It is then grasped by a small surgical forceps just below the point at which I wish to sever the upper portion from the lip. This excision above the forceps, however, is made after the frenum has been dissected out of the gum and from between the alveolar sockets of the central incisors as far down as the lancet will go, first on the right side and then on the left, while the frenum is drawn gently but firmly upward and outward with the forceps. This releases the frenum from between the teeth up to the point of normal attachment, and by stretching the frenum rather firmly and making the oblique incisions as deep as possible, the white fibers in the frenum are severed deeply enough to draw the labial portion out of the suture and permit the shrinkage and contraction of the tissue on the lingual side to draw out the lingual ends of the severed fibers. I have always believed that the fibers were thus eliminated either as a factor to maintain separation or to continue actively to cause it through the activity of the muscles of the lip to which the frenum is attached. I have performed this operation, and at his own wish permitted the patient to go directly from my office to school. The wound is usually quite well healed in two or three days. Most dental oral surgeons place one or two stitches in the inside of the lip where the triangular portion of the frenum is severed from the lip with a small pair of straight scissors. This cutting away of

the triangular portion and putting in the stitches (if they are to be employed) is the last step in the operation. I did not use sutures, merely painting the wound with iodine, a procedure which never resulted in any complications. Nevertheless, I think one or two small sutures on the inside of the lip is a better surgical procedure. The operation, after the anesthesia is secured, does not take more than three or four minutes, perhaps a little longer if stitches are put in.

To any dentist, and especially any orthodontist, a ruptured frenum is a rather common sight. Few children pass through the first two or three years of their life without getting their share of falls, very often striking the face, and resulting many times in a rupture of the frenum to a greater or less distance from the summit of the alveolus. In only a few instances where I have noted this, were the parents able to remember when it had occurred, though they did occasionally remember it quite definitely. Of course, when the frenum is completely normal in its attachment, it is unlikely that any except the severest blow or fall could rupture it; but, where it is bound down tightly to the summit of the maxillary dental arch, a smart blow on the upper lip in an upward direction will often rupture it to a greater or less extent. Such an accident does not help the situation in a truly abnormal frenum but rather complicates the operation for correcting it. It leaves a little pointed flap hanging down from the inside of the lip while the lower portion of the frenum lies on the labial gum tissue. It is usually darker in color and, fractured or unfractured, is unsightly and unsanitary when it is definitely abnormal. I have found such cases still maintaining separation of the teeth even though the frenum had been ruptured sufficiently to eliminate the muscle pull from side to side, which is further evidence to my mind that in many cases the frenum does pass through the premaxillary suture. Such a case is more difficult to operate.

As to the best time for correcting the abnormal frenum. Many years ago I believed it should be done as early as two years. In one case, I distinctly remember, the mother was a better orthodontist than I. I saw her child at the age of about one year and told her the sooner the frenum was corrected the better. It has not been done yet. The child has a thick, meaty, frenum today, but the attachment is about at a normal point on the anterior labial wall of the maxillary arch which developed downward and away from the attachment. This cured my enthusiasm for very early operations.

Then I thought the best time was just after the maxillary deciduous central incisors were lost and before the permanent ones were erupted enough to be an interference to the operation. Actually, I believe most cases can be diagnosed as normal or abnormal at this time, but I have found the operation quite successful at a later date; and, if one prefers to be ultraconservative, I think there is no great harm in putting it off for a year or two longer, though I believe it is done better at about the time nature is shifting from the deciduous to the permanent teeth. There is also a more open field for operating at this time than after the permanent central incisors have fully erupted.

I never had any complications in any of the perhaps fifty or seventy-five cases which I did myself nor in any of an equal number which I have referred to exodontists since I discontinued doing the operation myself. I cannot find

any ground to condone the attempt to correct the abnormal frenum by pinching it off through a long orthodontic treatment when it can be done definitely and decisively and to my mind far more effectively in a three to ten minute operation which will be healed completely in a few days and which has never left any visible scar in the many cases which have passed through my hands. On the contrary, the fleshy, red, unsightly, abnormal frenum is truly disfiguring. When properly removed the gum tissue is as even both in color and in contour as in patients who never had and did not need the operation.

In the last month or two, since beginning this paper, I have observed several patients who were operated on two years or more ago. Even a dentist could not have told that an operation had ever been performed, as there was no scar either on the labial gum or under the lip. These cases had had a couple of sutures placed on the area where the triangular portion of the frenum had been excised from the undersurface of the lip.

In conclusion it is well to point out that because all individuals have a frenum, normal or abnormal, the patient must depend entirely on the honesty of the doctor as to whether his case needs correction or not. Although I am not aware of any definite statistics as to their incidence, my own feeling is that if an orthodontist finds that he is operating for abnormal frenum on more than 3 or 4 per cent of his total number of patients, he is allowing his enthusiasm to influence his judgment.

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833 PARK AVENUE

CONTROL OF INFLAMMATORY CONDITIONS OF THE ORAL MUCOSA DURING ORTHODONTIC TREATMENT

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THE progress and accomplishments of that specialty of dentistry which has for its object the prevention and correction of dental and oral anomalies are a source of gratification to both the orthodontist and the general practitioner. A common meeting ground is found in the effort to initiate orthodontic treatment where and when indicated and in maintaining the integrity of the structures so carefully developed.

The orthodontist is charged with the treatment of conditions arising from aberrations of growth and function; the general practitioner assumes the responsibility of treatment of dental caries and diseases of the supporting structures of the teeth and the oral mucosa. In discussing the subject, the term gingivitis has been used in a broad sense, and it also has been assumed that gingivitis is a common ailment, affecting a majority of child patients whether undergoing orthodontic treatment or not. The relation to the orthodontic problem is found in the complications introduced by regulating appliances.

That the ultimate well-being of the periodontium is seriously threatened by the presence of the acute and chronic inflammatory conditions is clearly revealed by a brief survey of the pathology of gingivitis. The normal protective epithelial covering of the gingiva becomes stretched and thin; due to hyperemia and swelling of the underlying connection tissues. The flow of blood in the peripheral vessels (capillaries and venules about the gingival margin) is slowed; phagocytosis and lymph exudate are noted in the inflamed tissue, while active hyperemia in the deeper tissues remains. As a subjective symptom the presence of an increased blood supply in the deeper tissues is manifested by a feeling of tooth elongation and pain to biting pressure.

If these degenerative changes were confined to the soft tissues only, permanent and irreparable damage would not occur so frequently, but gingival irritation often progresses to the alveolar crest bone, followed by hyperemia which causes such degenerations as decalcification and osteoporosis.

Decalcification of the alveolar crest bone results in permanent injury to the alveolar crest and transseptal fibers of the periodontal membrane, increased depth of the gingival crevice and eventual pocket formation.

Clinical experience proves that early recognition and prompt treatment based on a careful consideration of known etiologic factors pay generous dividends in terms of normal gingival structures.

The orthodontist may choose only to institute the ordinary measures of preventive treatment which can be done easily during office visits of the patient, thoroughly polishing the teeth and appliances at regular intervals, using a

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rubber cup and a paste of pumice, to which has been added a small amount of sodium perborate. As much of the appliance should be removed as is convenient and the cleaning paste forcibly pumped into areas difficult of access. Sodium perborate and hydrogen peroxide have little value as to their bactericidal properties, but are valuable in disorganizing and removing organic debris. This is followed by a nonirritating antiseptic forcibly applied with a rubber cup.

Occasionally there is found a child patient peculiarly susceptible to recurring infections of the fusospirochetal type. These unfortunate children tax the patience and skill of the orthodontist and the general practitioner, and demand cooperative effort, to be discussed later, for success in treatment.

For convenience in discussion, it may be assumed that, excepting the precautionary measures spoken of, the general practitioner will be charged with the greater responsibility in maintaining the health of the mouth during orthodontic treatment.

He must, then, hold to the general principles underlying the diagnosis and treatment of any disease, carefully seeking the cause and proceeding systematically along the lines suggested by the known etiologic factors.

It is convenient to divide oral disease into two classes, based on (a) systemic factors, and (b) local factors. There is a group of grave systemic diseases which frequently manifest their primary lesions about the oral mucous membranes. It is not supposed that by training or experience a dentist is qualified to make a diagnosis of systemic diseases by their oral manifestations. He should, however, regard with suspicion any morbid condition that does not respond to treatment within a reasonable time limit, and refer the patient to his medical adviser. An ounce of consultation often may save a pound of grief.

Some constitutional diseases with oral manifestations are: measles, scarlet fever, chickenpox, lichen planus, pemphigus, erythema multiforme, diphtheria, syphilis, tuberculosis (secondary invasion).

Blood dyscrasias include: agranulocytic angina, pernicious anemia, hemorrhagic purpura, leucemia.

Dietary deficiencies: quantity, and quality (lack of specific vitamin and mineral elements).

Endocrine imbalance; particularly diabetes, which is due to lack of insulin. While it is exceptional to find hypoinsulinism in young patients, I have cared for mouth lesions of three children afflicted with diabetes. Until the urine is free of sugar and the blood sugar is within normal limits, gingival disease stubbornly resists treatment, and surgery should be avoided if possible.

Fortunately, most of the blood dyscrasias are rare diseases, and it is doubtful if many dentists will encounter such complications; but, when the entire list of possibilities is considered, the systemic background of oral disease is an important potential factor.

The local etiologic factors of diseases of the oral mucosa are well known: (1) mechanical irritants, (2) teeth in abnormal position, (3) lack of function (disuse), (4) trauma due to excessive or abnormal stress in functional occlusion, (5) infection.

Mechanical irritants are: improperly contoured fillings, overhanging margins of fillings, in fact poorly conceived and executed dental restorations of

any kind. This is not an orthodontic problem, but it may become a problem for the orthodontist who is compelled to suggest the obvious treatment to the general practitioner.

Teeth in abnormal position are responsible for areas which retain food, particularly in the child patient who goes in a big way for ice cream, pastries, candies, and mucilaginous foods of like nature. In the child undergoing orthodontic treatment, the tooth will assume a normal position, which is the orthodontist's responsibility; but sometimes it requires the united efforts of the general practitioner, the orthodontist, the parents, and the nurse to induce the child to take the proper food and use the necessary care in removing residual food after eating.

Lack of function may be the result of pain due to untreated carious cavities, or an existing periodontal inflammation. Chewing habits, which are difficult to correct, are formed to favor the painful teeth. As the stimulus of function is necessary to the normal development of the dental structures, it is important that the cause of disuse be established and corrective treatment instituted.

At certain periods during orthodontic treatment, unavoidable inharmonious occlusal relationship may be produced. The resulting inflammation usually is transitory in nature, and may be controlled by counterirritation and subgingival drug therapy, the latter to raise the threshold of resistance to infection.

Infection: Kirkpatrick[1] neatly summarizes infection as follows: "Infection of the gum tissue will occur, providing (a) the infecting organisms are present in virulent form, and (b) the tissues are so degenerated as previously described by injury and inflammation that (1) their defense is lost and (2) they can be readily reduced by oxygen, thus rendering favorable pabulum for anaerobic organisms. The two factors, degenerated epithelial tissue and infecting organisms, must be present before infection can occur. The severity of the infection depends on the virulence of the organisms and the degree of catabolism in the tissues. Once infection becomes established (and it is usually fusospirochetal in nature) inflammation sets in."

In spite of the great amount of work done on the fusospirochetal infections, as evidenced by a voluminous literature, it is not definitely proved that these organisms are the causative agents. The constant association of the fusospirochetal group with inflammatory conditions of the oral mucosa, however, leads to the belief that in spite of definite proof they are the chief factors in oral and gingival infections.

Most pathogenic mouth organisms are anaerobes, which suggests a fundamental principle underlying all treatment.

Office treatment is conveniently divided into three phases:

(1) Changing the environment of the organisms by the elimination of such areas as offer favorable fields for anaerobic activity. In this category is found increased depth of the gingival crevice due to hypertrophy of the gingiva, pericoronal flaps over erupting teeth, inaccessible spaces formed by fillings, crowns, or appliances.

(2) Mechanical removal of calculus and food deposits. Particular attention to oral hygiene is necessary. The factor of food stagnation is important, as

aerobic bacteria become active in these areas, exhausting enough oxygen to render the deeper field favorable to anaerobic growth. It is possible to reduce the oral bacterial count by scaling and polishing the teeth, at the same time reducing the possibility of colony growth in protected areas.

(3) Drug therapy. Whether infection is primary or secondary, the value of drugs as a supportive measure in time of stress is established. Although there are few specifics in medicine, drugs may be selected for a specific purpose with much better effect than is obtained by closing the eyes and letting fly with both barrels, hoping for general results.

Oxygen liberating compounds such as sodium perborate and hydrogen peroxide serve the double purpose of thorough débridement and the introduction of oxygen into inaccessible areas, thus discouraging bacterial activity.

Antiseptic drugs are legion, and their efficiency depends more on intelligent selection and manner of application than on any inherent magic quality. Nonirritating drugs may be combined with a polishing paste as a routine procedure; this not only saves time, but insures intimate contact and prolonged exposure of the organisms to the drug. Rotation of drugs occasionally is necessary, as repeated applications may result in an increased resistance of microorganisms to a particular remedy. For many years I have advocated the following home treatment for patients with stubbornly resistant or recurring fusospirochetal infections.

The patient is given an ampoule of 5 per cent salvarsan in glucose with instructions to brush the teeth thoroughly; then, using another brush which has been charged with the salvarsan solution, the teeth are again brushed, forcing the solution into the gingival crevice and interproximal spaces, and not following this with a liquid or food for some time.

The results of this treatment are always good, and sometimes spectacular. Peculiarly susceptible patients, who suffer frequent recurrences, often are kept in a state of equilibrium by this preventive measure. No manifestations of arsenical overload have been observed over a period of many years.

Escharotic agents. Ordinarily the use of tissue-destroying drugs is not indicated, but hypertrophic or hyperplastic gingivitis demands their use; and when reasonable care is exercised, good results may be effected.

The general practitioner should work in harmony with the orthodontist, surmounting the obstacle of regulating appliances by various expedients. Usually success is the reward of patient effort, but occasionally there comes a sad day when conservative methods fail, when disease gets the upper hand, and it is believed that removal of the appliances is necessary. Having in mind the physical welfare of the patient and the necessity of assurance of the eventual integrity of the periodontium, the orthodontist and the general practitioner must arrive at an agreement. The general practitioner must not be swayed by expediency; the many hours necessary to construct and apply regulating devices must be carefully weighed; and in a like manner the orthodontic mind must be prepared to yield, if necessary, to the suggestion of a necessary measure. If appliances are removed, treatment should proceed as rapidly as possible so that the interruption will not prove a serious handicap to orthodontic treatment.

SUMMARY

Acute and chronic inflammations are responsible for permanent damage to the periodontium.

A high percentage of child patients are peculiarly susceptible to gingivitis. The patient undergoing orthodontic treatment differs only in the complications introduced by appliances.

Early recognition and prompt treatment of oral lesions pay large dividends in terms of mouth health.

The responsibility of maintaining optimum oral health during orthodontic treatment should be divided between the orthodontist and the general practitioner.

REFERENCE

1. Kirkpatrick, R. M.: The Aetiology of Gingivitis, Dental J. Australia, March, 1936, p. 141.

SUPERNUMERARY TEETH

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SUPERNUMERARY teeth have for a long time been recognized as one of the direct causes of malocclusion. Perhaps on account of this being so obvious, or because correction of the resulting deformities are apparently simple, a study of the available orthodontic literature on this subject will reveal that comparatively little effort has been spent on this problem as compared with other more or less important ones. This fact was forcibly brought to my attention when a girl came to our office with the dental deformity as illustrated in Fig. 1. This patient had been allowed to reach the age of eighteen before seeking orthodontic assistance of which there is now considerable need; whereas, five or ten minutes' work on the part of her dentist years ago would have prevented her disfigurement and needless expense and embarrassment.

It is my purpose in this paper to discuss in a simple manner the attributed etiology, diagnosis, classification, and treatment of the supernumerary tooth and its resulting problems from the viewpoint of the orthodontist.

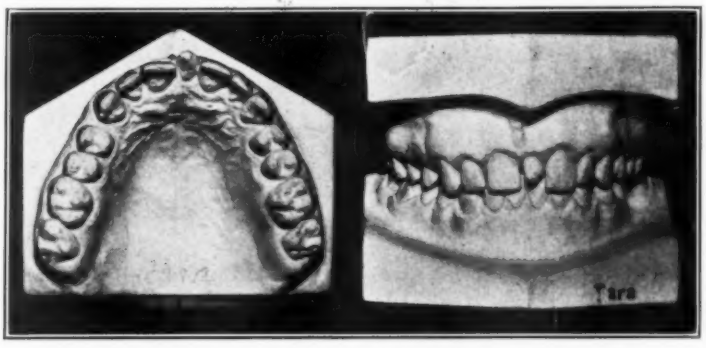

Fig. 1.—Result of neglecting a supernumerary tooth.

The etiology of the supernumerary tooth is not yet clearly understood and even if known would probably help but little in our problems concerning the extra tooth, unless perhaps we could choose our ancestors and control precisely our development from the time of the union of the male and female gametes. Many have attributed, and still do attribute, the presence of these unwanted teeth to atavism—their theory being that they are an indication on the part of nature to restore in man teeth which have been lost in the process of evolution. Much could be said in support of this theory. Enough case histories have been recorded to show that heredity undoubtedly plays some part in their presence, but investigators have found it quite difficult to tract supernumerary teeth in ancestors perhaps because their existence has been unknown or unnoticed.

Man, at one time, had forty-four teeth in his dentition. The course of evolution has dropped twelve teeth. Supernumerary teeth in nearly every instance

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are located in the dental arch erupting or trying to erupt in a position that was formerly held by one of the teeth dropped by the dentition in the course of time. This seems of some significance.

Many scientists have rejected the theory of atavism, claiming that the presence of extra teeth may be more satisfactorily explained as being due to other causes. Based on embryological studies the most satisfactory explanation is one which Dr. G. V. Black supported. Briefly stated, the theory is this: The important structure in the origin of a tooth is the enamel organ, since it is the last rudiment to appear and its presence conditions the formation of the dentinal papilla. The size and form of the tooth are dependent upon the size and form of the enamel organ. The epithelial cord, after having preceded the enamel organs of the permanent teeth, normally disappears. It may, however, fail to be resorbed completely following the closure of the follicle, and epithelial remnants may persist. If large enough, these remnants, or epithelial whorls as they are called, are capable of exciting and controlling the development of a dentinal papilla, provided they occur in a field fertile for development. If the proliferations of the epithelial cord are normal in number, we may expect the

Fig. 2.—*A*, Case treated without roentgenograms; *B*, after treatment.

dentition to develop in a normal manner. We have the embryologic evidence that abnormal proliferations do occur. The extent of their development is probably related to their position in the jaw. If situated deeply, in or near the position of those which give rise to normal teeth, we may expect to find "duplicate" teeth or supernumeraries similar in shape and size to the normal. If located too close to the normal germs, they may become fused in the process of their development and give rise to the various kinds of fused, "twin," or partially divided teeth. If situated too far laterally, they may receive insufficient nourishment and become "peg" or conical teeth. If located too superficially, they may entirely fail to develop. If the germs are very small in size they may develop into so-called "denticles." Compound composite odontomas, multiple-coned teeth, transposed molars, etc., are undoubtedly due to abnormal development of the enamel organ of a tooth germ.

Diagnosis of Supernumerary Teeth.—The prime requisite in the diagnosis of supernumerary teeth is a good and complete roentgenogram of the case. This may be well shown by the case illustrated in Fig. 2.

This case was treated about twenty-five years ago by an orthodontist who, in spite of his best efforts over a considerable period of time, was unable to make

any appreciable change in the position of the teeth. X-ray pictures, a relatively novel procedure at that early date, disclosed two long, well-developed supernumerary teeth which proved to be the cause of his troubles. Their removal permitted the concluding of the treatment in the fairly satisfactory manner as illustrated.

Supernumerary teeth that are exposed to view are usually readily distinguished from the adjacent normal teeth by their variation in form, size, and position. Frequently, however, especially in the incisor area, the supernumerary may be identical to its adjacent tooth, and good judgment must be shown in the selecting of the least desirable tooth if removal is indicated.

In studying roentgenograms for evidence of supernumerary teeth care must be taken to avoid misinterpretations of the films. Improper technique, accidents, and certain pathologic conditions may give the appearance of a supernumerary. Dr. Clarence O. Simpson points out the density of the supernumerary tooth, the trace of an enamel cap, longitudinal streaks corresponding to the location of the pulp canal, and the radiolucent line of demarcation of the remains of the tooth sac as contributory evidence in the identification.

Fortunately orthodontists in roentgenographic diagnosis are seldom confronted by the problems which decalcifying supernumerary teeth present, as this condition has seldom (never to my knowledge) been observed in patients under thirty-five years of age.

Morphologic Variations.—One of the most interesting aspects in the study of the supernumerary teeth is the variety of shapes and sizes which they assume. Often they assume the form of the normal teeth in the region in which they erupt; more often, however, they exhibit imperfect forms with single or multiple cusps, but usually single rooted. For ordinary diagnostic purposes, they may be simply classified as follows:

1. Teeth normal in form and size.
2. Teeth normal in form; abnormal in size.
3. Conical or "peg-shaped" teeth.
4. Teeth exhibiting irregularly shaped crowns with several cusps.
5. The so-called "pin" teeth as found in compound composite odontomas.
6. Teeth fused to normal teeth.

(1) In Fig. 3 is illustrated a case in which the supernumerary lateral incisors (supernumerary teeth which resemble normal teeth in form usually take their name from the adjacent tooth which they resemble in appearance) were of normal size and shape. In this case the supernumerary teeth were extracted before the permanent canines were terribly deflected, and the dentition developed in a normal manner.

These duplicates may occur anywhere in the mouth but usually are located in the anterior segment. Sometimes the differential diagnosis presents great difficulties, although in considering it from the treatment angle, one should always remove the tooth whose absence will most facilitate treatment. Supernumerary teeth in the premolar area are usually duplicates in form of the normal teeth of that region but they are comparatively rare. As many as ten super-

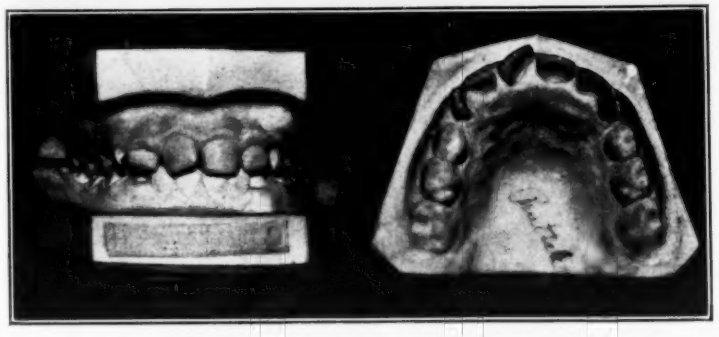


Fig. 3.—Supernumerary teeth normal in form and size.

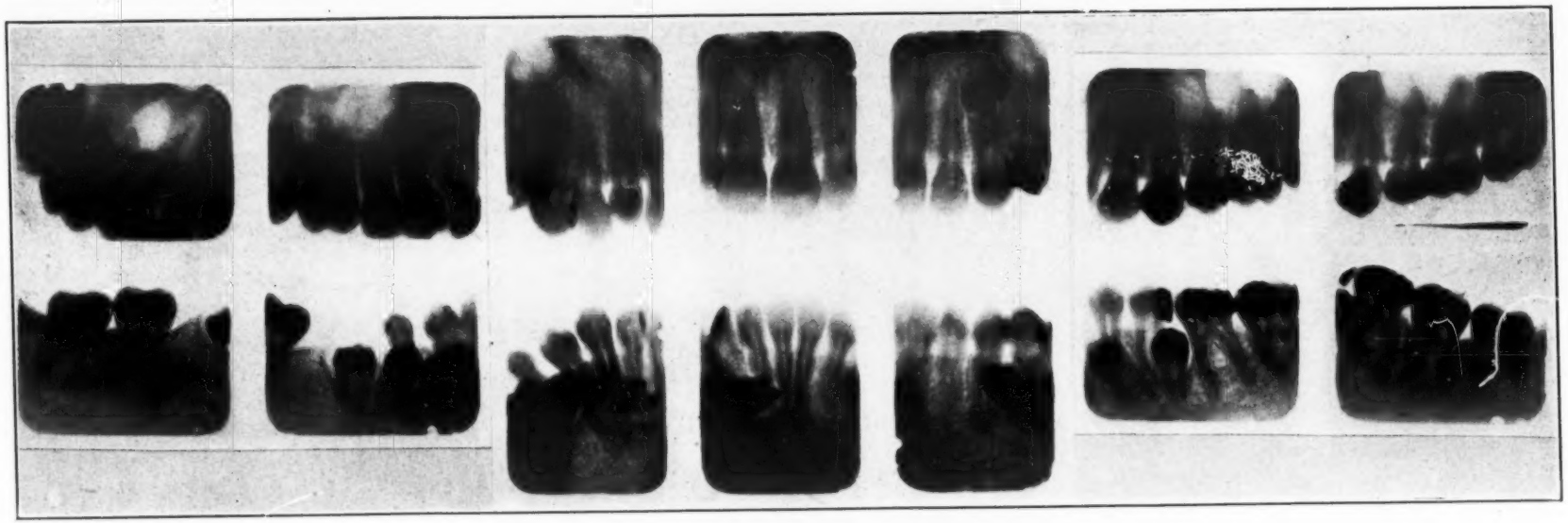


Fig. 4.—Numerous supernumerary premolars.

numerary premolars of comparatively normal form in the same arch have been reported. Fig. 4 shows a case with numerous supernumerary premolars. Note their apparent normality in size and shape.

(2) Teeth normal in form, abnormal in size. Statistics show that this group is also usually found in the anterior part of the mouth although occasionally it may be found as premolars and molars. Fig. 5 illustrates a supernumerary lateral incisor attached to the model after its removal. This group is quite common.

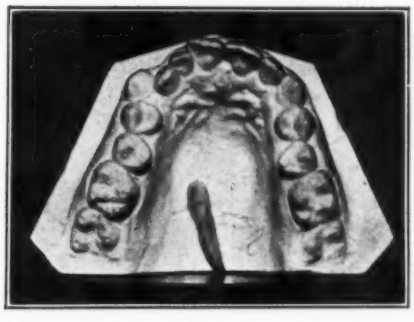


Fig. 5.—Supernumerary tooth normal in form, abnormal in size.

Fig. 6.—Deciduous conical or peg tooth.

(3) The conical or so-called "peg" tooth is probably the commonest form of the supernumerary tooth. It is usually found in the anterior part of the maxilla and causes a great variety of malpositioning of the individual teeth. Fig. 6 illustrates an interesting case of a cone-shaped deciduous supernumerary tooth. The appearance of such a tooth after the shedding of the deciduous central incisor gave the little patient's parents cause for alarm until the x-ray examination revealed it as a supernumerary tooth. Dental literature records deciduous supernumerary teeth as being scarce. Of the fifty odd cases presented at our office during the past ten years, but two have been in the deciduous set. Dr. Carrea of Buenos Aires reported a very interesting case of two deciduous supernumerary incisors being replaced by supernumerary incisors in the permanent dentition.

Fig. 7 illustrates a cone-shaped supernumerary tooth lingual to the maxillary right central incisor as it was in situ.

The conical form is seldom found in the premolar area.

(4) Teeth exhibiting irregularly shaped crowns. The supernumerary teeth in this group are present in some of the most unsightly and disfiguring cases as

illustrated by Fig. 8 *A*. Fig. 8 *B* shows an irregularly shaped supernumerary lying between a maxillary lateral incisor and a maxillary central incisor as shown by the x-ray picture in Fig. 8 *C*.

(5) The so-called "pin" teeth as found in compound composite odotomas. Fig. 9 shows a case with the "nest" of teeth in the approximate position in which they were found by the exodontist. Twenty-two miniature teeth of various shapes and sizes and of varying degrees of development were found in this cyst. After its removal the miniature teeth were placed in a roll of paraffin moulded to the form of the cyst and located in the impression before the plaster was poured. It took approximately two years for the permanent left central incisor to assume its place in the arch. These conditions are relatively rare but are most interesting. Some of the teeth were most minute in size; some almost perfect miniatures of normal teeth.

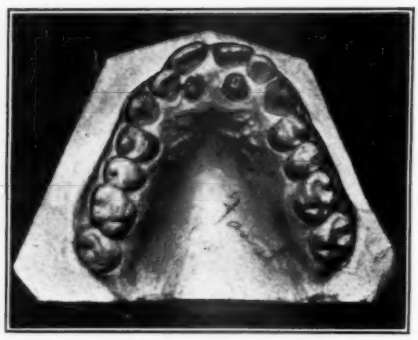


Fig. 7.—Cone-shaped supernumerary tooth.

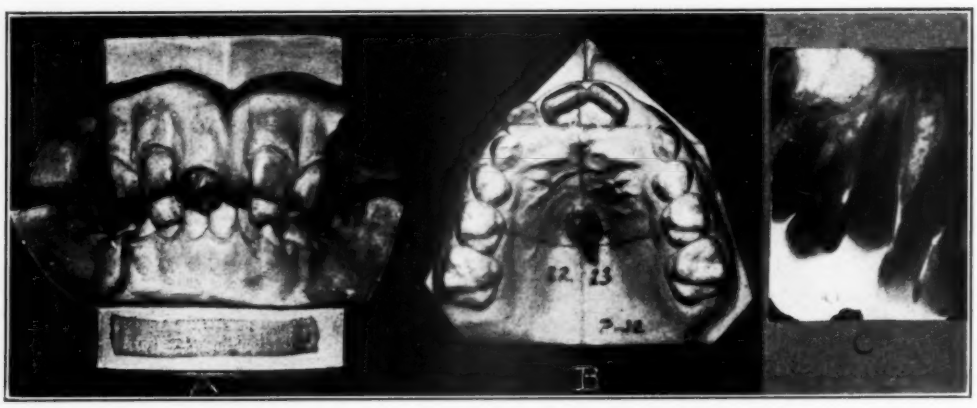


Fig. 8.—Irregularly shaped supernumerary teeth.

(6) Teeth fused to normal teeth. Most of the cases falling in this group that have been recorded in dental literature are described as fourth molars fused to third molars. This seems to occur rather frequently, but fortunately they are of little interest to the orthodontist because of their position and time of eruption in the dental arches. Occasionally, however, a supernumerary is found fused to an anterior tooth, a condition which sometimes may present an extremely difficult problem as shown in Fig. 10. I shall discuss the treatment of a similar condition later in this article.

Supernumerary teeth may be fused to each other as well as to normal teeth. Fig. 11 shows such a condition. Here an irregularly shaped one is found fused to a "peg" shaped one in the root portion. Their presence caused the impaction of the central incisor. The x-ray picture did not clearly show the union.

Before discussing the classification and treatment of malocclusion caused by the supernumerary tooth, it may be worth while to consider some of the other complications which they may cause. Simpson has stated that supernumerary teeth occurring in the region of the maxillary incisors are not usually important clinically, and in the absence of symptoms should be disregarded. Stafne points out that in view of the relatively large number which became cystic it is important that periodic radiographic examinations be made so as to prevent possible extensive loss of teeth, involvement of the maxillary sinus, and

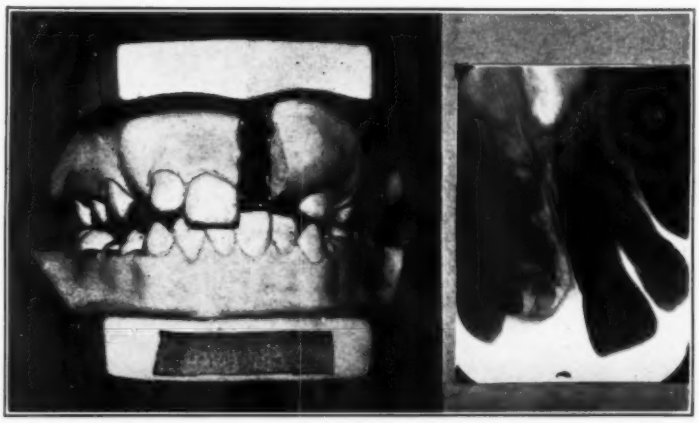


Fig. 9.—Compound composite odontomas.

Fig. 10.

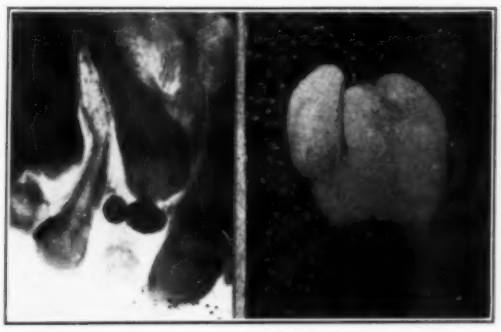


Fig. 11.

Fig. 10.—Supernumerary teeth fused to central incisors.
Fig. 11.—Fused supernumerary teeth.

other complications. Observers have found that approximately as many supernumerary teeth are found in an inverted position as are inclined normally. There is a tendency for these teeth which are inverted to erupt toward the nares. It is possible that this approach to eruption and partial eruption of these teeth in the floor of the nares can be responsible for chronic irritations of the mucous membrane and for other sequelae. Charleroi reported a case in which a supernumerary maxillary central incisor had erupted into the nostril of a girl nine

years of age, and had caused abundant secretion and nasal obstruction. Long reported the case of a youth, eighteen years of age, in whom such a tooth was responsible for an opening from the oral cavity to the nasal cavity, and there was a regurgitation of fluids into the floor of the nares. Dr. P. Gaugery reported a case in which a severe pain at the base of the nasal bone was proved to be caused by a supernumerary tooth. In the region of the third molar a supernumerary tooth may have a deleterious effect on the life and health of the normal teeth. Fig. 12 shows a supernumerary molar whose presence caused pressure absorption of the distal root of the second molar, manifested this action by severe pain in this region and resulted in the loss of both the second and the third molars.

Fig. 13 *A* and *B* illustrates the horizontal impaction of the mandibular third molars occasioned, no doubt, by the presence of the supernumerary fourth molars. Fig. 13 *C* shows the perpendicular impaction of a mandibular third molar due to the presence of an odontoma. Cases of this type are problems for the exodontist rather than the orthodontist. These roentgenograms of supernumerary molars were borrowed from the files of Dr. C. R. Grissinger of Pittsburgh.

Classification and Treatment of Cases Caused by Supernumerary Teeth.—The purpose of a classification is to place into groups cases which exhibit similar characteristics and thus facilitate the planning of the treatment. Supernumerary cases may be classified in various manners, but on this basis it seems logical to divide such cases into two main groups:

I. Simple, those cases characterized by the presence of one or more supernumerary teeth which have caused the malposition of proximating teeth.

II. Complex, those cases characterized by the presence of one or more supernumerary teeth whose presence has caused malrelation of the dental arches or the malposition of the majority of the teeth in one arch. To this group also must be added those cases in which the supernumerary merely complicated an existing malformation.

I. Simple.—In this group fall the many and varied types of malocclusion which are expressed in definite locations in the arches. A more or less complete list of this group with a brief discussion of their treatment follows:

A. Labial, buccal, lingual displacement of normal teeth by supernumerary teeth.

B. Mesial or distal displacement of normal teeth by supernumerary teeth.

C. Torsal displacement of normal teeth.

D. Transposition of normal teeth.

E. Impaction of normal teeth.

F. Supernumerary teeth in line of occlusion.

G. Fusion of supernumerary teeth to other teeth.

H. Absence of normal teeth, presence of supernumerary teeth.

I. Combinations of the simple cases.

II. Complex—In this group are those cases characterized by malrelation of the dental arches to each other or by malposition of groups of teeth.

A. Broadening of the entire dental arch through the presence of supernumerary teeth.

B. Cases in which the positioning of the arches was influenced by the presence of supernumerary teeth.

C. Existing malocclusions complicated by supernumerary teeth.

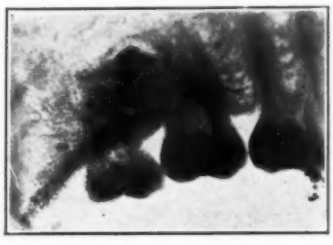
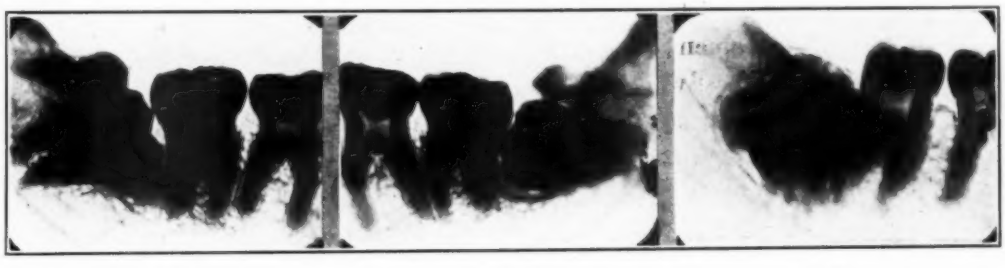


Fig. 12.—Supernumerary molar causing pressure absorption.



A. *B.* *C.*

Fig. 13.—*A* and *B*, Horizontal impaction of third molar by supernumerary tooth; *C*, perpendicular impaction by odontoma.

Fig. 14.—Labial displacement of central incisor by supernumerary tooth.

I. Simple.

A. Labial, buccal or lingual displacement of normal teeth by supernumerary teeth. Fig. 14 is an example of this type. The presence of the left supernumerary lateral incisor has displaced the left central incisor labially. Treatment for this type of case as well as for all the other types of simple cases was capably outlined as early as the seventh century by Paul of Aegina (625-690), the last classical Greek scholar, who was the first to consider: "When supernumerary teeth cause an irregu-

larity of the dental arches, they may be corrected by resection of such teeth or by extraction.'' This anciently prescribed treatment is still the solution for most supernumerary cases today followed by judicious orthodontic procedures. If the supernumerary teeth in such cases as this are discovered at an early age, other things being normal, no treatment other than the extraction will be found necessary. The natural forces of occlusion will develop the malposed teeth into their normal place in the arch.

B. Mesial or distal displacement of normal teeth by the presence of supernumerary teeth. Fig. 15*A* and *B* illustrates a case in which the maxillary central incisors are displaced distally due to the presence of two supernumerary teeth.

The successful treatment of this case, Fig. 15*C*, was accomplished by the extraction of the offending teeth and developing the central incisors toward the median line in such a manner as to provide for their proper axial positioning. The lateral incisors were likewise moved mesially to approximate the central incisors, and the canines erupted in their normal position.

C. Torsal displacement of normal teeth by the presence of supernumerary teeth. A relatively large number of the simple cases to be found are of this type. One may well suspect the presence of a supernumerary if a case presents exhibiting the characteristics of the left central incisor in Fig. 16. Fig. 17*A* and *B* illustrates the torsal version of right central incisor and lateral incisor due to the presence of the supernumerary, which was waxed to the palate of the model after its extraction. The affected teeth were then developed mesial and rotated into position as illustrated. Fig. 18 illustrates a case of a girl nine years old showing the simultaneous eruption of the left central incisor and supernumerary. This happens quite frequently and extraction is indicated. This case is developing normally without treatment.

D. Transposition of normal teeth by supernumerary teeth. Fortunately this type seldom occurs, but when it does some of the cases may be quite complex from the treatment angle. Fig. 19 illustrates a case in which both central incisors are transposed by the presence of two irregular supernumerary teeth.

A survey of this case would show the lateral incisors approximately in their normal position. After extraction of the offending supernumerary teeth, the central incisors were carefully developed toward the median line. Eighteen months after starting treatment the central incisors were in contact, in fact slightly overlapping due to the loss of a mandibular first molar and the resulting underdevelopment of the mandibular arch.

E. Impaction of normal teeth by supernumerary teeth. This is one of the commonest forms of malocclusion caused by supernumerary teeth. In any case presenting with delayed eruption of a tooth or teeth, one may suspect the presence of supernumerary teeth. Fig. 20*A* illustrates the impaction of both permanent central incisors by two small supernumer-

Fig. 15.—Distal displacement of central incisors by supernumerary teeth. *A* and *B*, before treatment; *C*, after treatment.

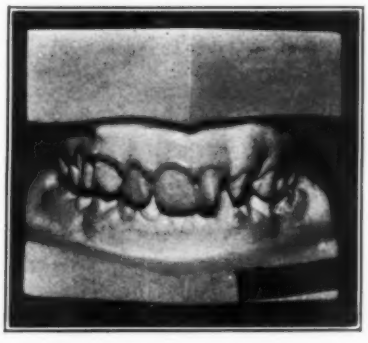


Fig. 16.—Torsal displacement by supernumerary tooth.

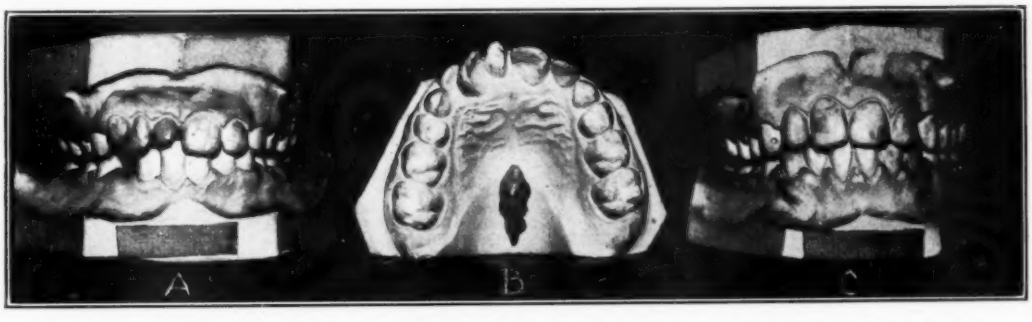


Fig. 17.—*A* and *B*, torsal version of central incisor before treatment; *C*, after treatment.

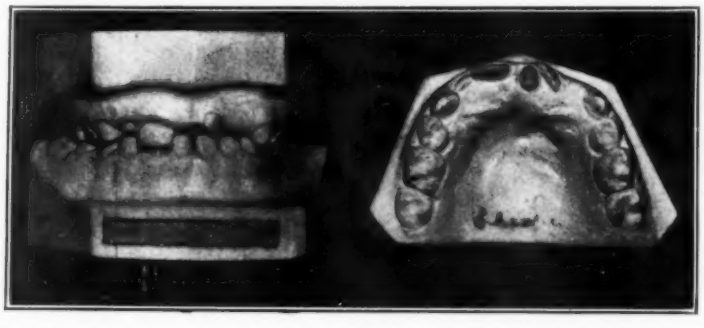


Fig. 18.—Supernumerary tooth erupting simultaneously with incisors.

ary teeth shown in situ as uncovered by the extractor. Fig. 20*B* shows the subsequent erupting of one of the central incisors four months later. Fig. 20*C* shows the end result several years later. The only orthodontic work instituted in the maxillary arch was some slight expansion in the anterior region. In the mandibular arch a horizontally impacted right lateral incisor necessitated considerable treatment.

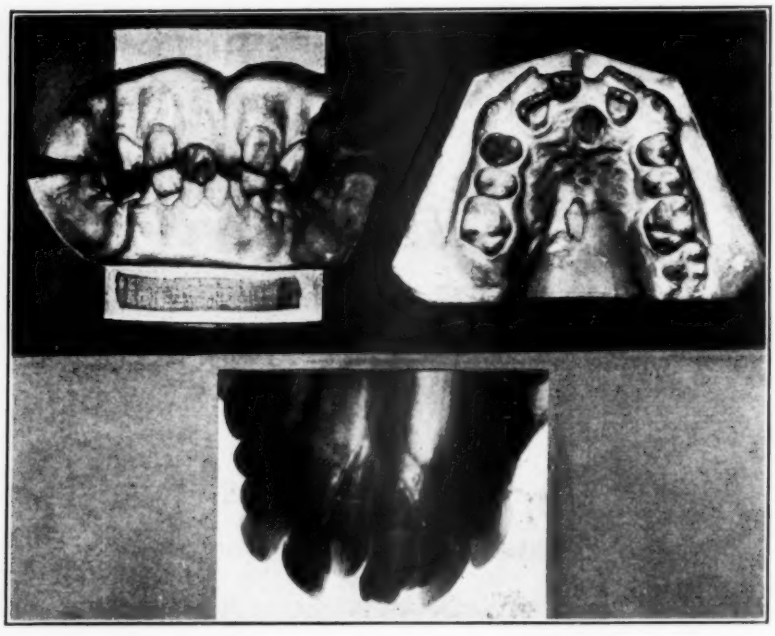


Fig. 19.—Transposition of incisors and supernumerary teeth.

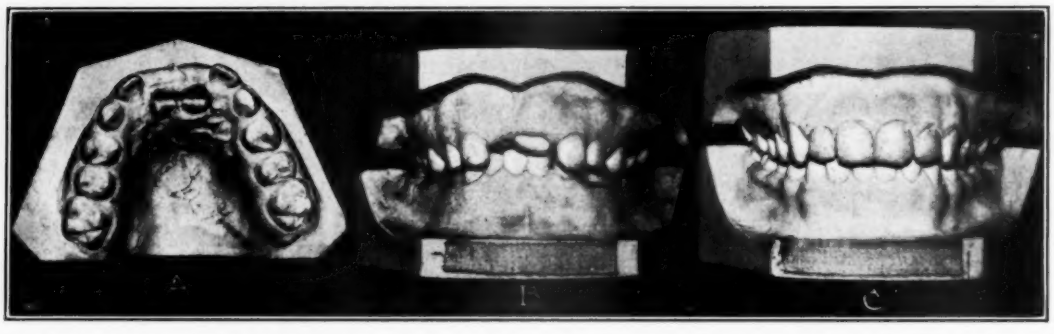


Fig. 20.—Impaction of central incisors caused by supernumerary teeth. *A*, supernumerary teeth in situ; *B*, four months later; *C*, after treatment.

Fig. 21*A* and *B* shows the impaction of a single central incisor by a supernumerary accompanied by another supernumerary which seemed to exert but little effect on the occlusion. They are shown in situ as found in their removal. Fig. 21*C* is a progress model made eighteen months later, at which time it was deemed advisable to assist the central incisor in its eruption because of its lingual inclination and apparent impaction. Accordingly, a lingual arch with an auxiliary spring was used to stimulate its proper development.

Similar cases are illustrated in Fig. 22, showing the extracted teeth in their approximate preoperative positions. No orthodontic treatment was found necessary in any of these cases.

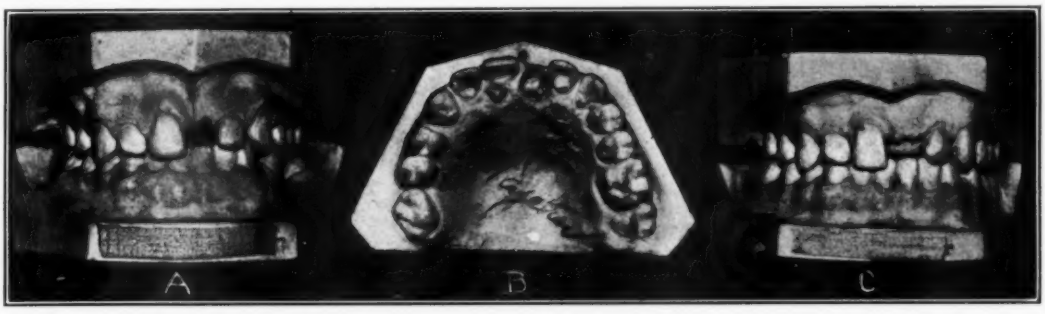


Fig. 21.—*A* and *B*, impaction of left central incisor by supernumerary tooth; *C*, condition eighteen months after extraction.

Fig. 22.—Impaction caused by supernumerary teeth requiring no orthodontic treatment. *A* and *B*, similar cases of maxillary right central incisor impaction; *C*, inverted supernumerary impacting central incisors; *D*, well-formed supernumerary teeth impacting central incisors; *E*, deciduous central incisors and supernumerary teeth in situ.

F. Supernumerary teeth in line of occlusion: This type is found rather infrequently but when presented is most interesting. Fig. 23 illustrates a case in which the presence of the supernumerary in line created but little disturbance. The central incisor, however, was extracted as shown by model, and a more perfect arch form obtained by rotating and aligning the mandibular anterior teeth.

G. Fusion of supernumerary to other teeth. Fig. 24*A* shows a model of a very interesting case of this type. The patient was a male, eight years of age. A supernumerary incisor was fused to the mesial surface of the maxillary left central incisor throughout the entire crown portion as shown by the roentgenogram, Fig. 24*B*.

The pictures show no apparent nerve anastomosis between the two teeth, and treatment was based on this fact. The dentist severed the coronal fusion with a disc, and then proceeded to extract the supernumerary. Fig. 24*C* shows condition as presented after the extraction and at inception of treatment. At this time the crowns of the incisors were in contact, and the appliance was changed to improve the axial position of the central incisor. There have been no clinical manifestations of any sort to condemn this rather radical treatment.

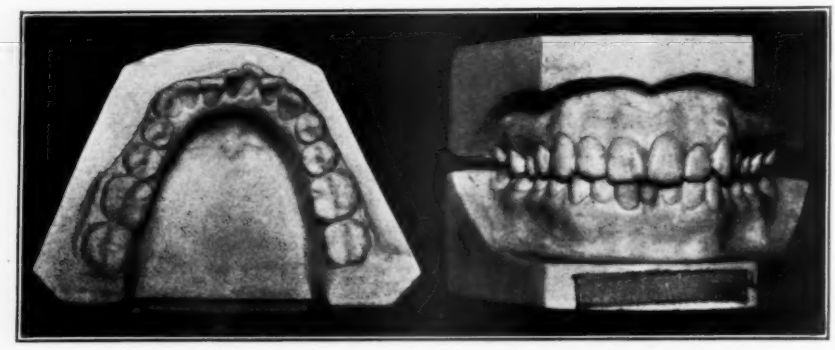


Fig. 23.—Supernumerary tooth in line of occlusion.

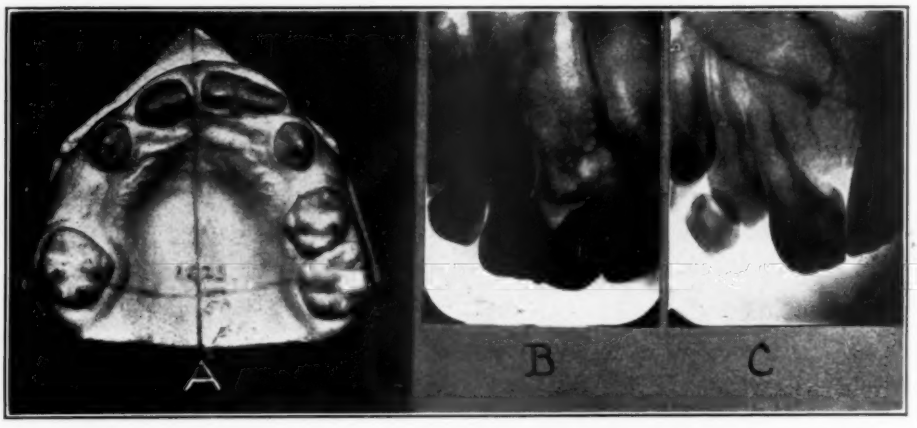


Fig. 24.—*A* and *B*, supernumerary tooth fused to central incisor; *C*, after extraction and inception of treatment.

H. Absence of normal teeth, presence of supernumerary tooth. I include this somewhat questionable group because it was suggested by the late Dr. Pullen, whose paper on the problems of the supernumerary teeth published in 1915 is the most complete and interesting one to be found in dental literature. He suggested such a group to include cases in which a normal tooth might be congenitally absent but a supernumerary peg-shaped tooth might be present to take its place. Of course, this statement leaves much room for argument. However, continuing on this supposition Fig. 25 shows a case in which the maxillary right lateral incisor may be missing and a supernumerary right central incisor is present in its place. It may be, however, that the right lateral incisor enjoyed phe-

nomenal growth and merely appears as a central incisor. While on this subject of oversized teeth I should like to show an unusual anomaly which was presented at our office. Fig. 26 shows a boy, six years of age, who had all deciduous teeth in place except the maxillary right canine, which had been replaced by a "whopping" big cuspid. Roentgenograms showed no further abnormalities in the dentition, which seems now to be developing quite normally. The x-ray picture banished my hopes for a super-supernumerary.

Fig. 25.—Supernumerary central incisor in place of congenitally missing lateral incisor.

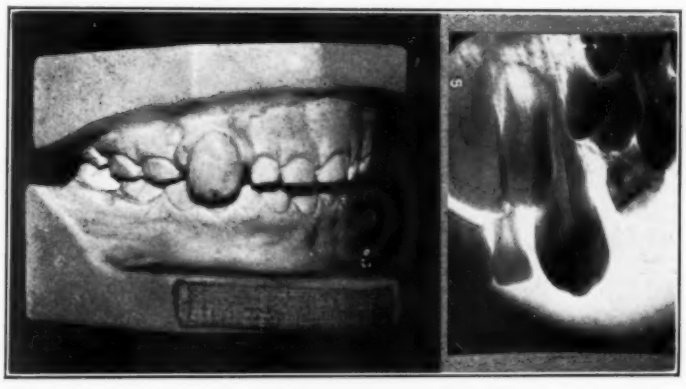


Fig. 26.—Canine anomaly.

I. Combinations of simple cases. This group is included to take care of such cases as shown in Fig. 27, which combines two or more of the previous groups. In this particular case is found the torsal version of the maxillary left central incisor as well as the impaction of the left lateral incisor by the presence of the supernumerary lateral incisor and the diminutive canine.

II. Complex.

A. Broadening of dental arch by presence of supernumerary tooth. Fig. 28 illustrates a case in which the entire anterior segment of the arch is widened by the presence of the supernumerary "peg" central incisor. Ideal treatment of this type of case is difficult inasmuch as it would be necessary to develop every tooth in the arch to the arc of a smaller circle while maintaining proper axial inclination. Much grief could be spared by early discovery of such a condition.

B. Relationship of arches influenced by supernumerary teeth. Fig. 29 shows a case exhibiting malrelation of the dental arches. This condition no doubt was greatly influenced by the presence of the supernumerary tooth which locked the mandible with each closure of the jaws in the most retruded position possible. This continual action through the developing period helped prevent the normal forward growth in the mandibular

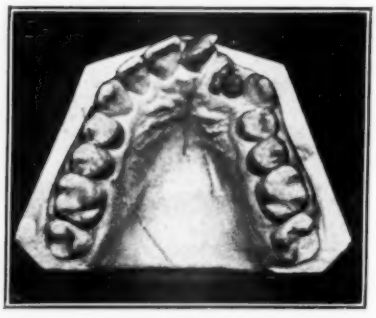


Fig. 27.—Combination of impaction and torsal version.

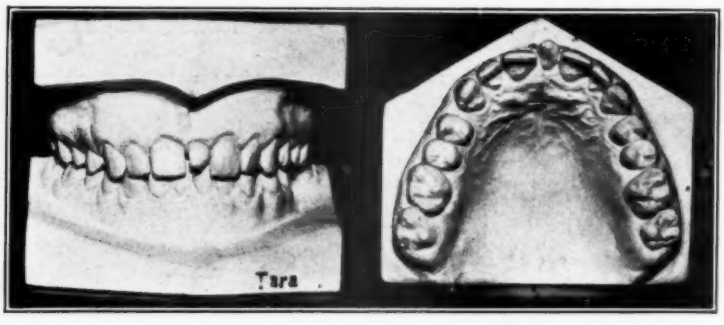


Fig. 28.—Broadening of anterior portion of arch by presence of supernumerary tooth.

Fig. 29.—Relationship of arches influenced by supernumerary tooth.

arch. Proof that this supernumerary tooth was a causative factor lay in the manner in which the case reacted after its extraction. The mandible which had been held in a retruded position moved forward and stayed forward with a minimum of treatment and effort.

C. Existing malocclusion complicated by presence of supernumerary teeth. This group includes those deformities exhibiting one or more super-

numerary teeth whose presence merely adds to the complication of the malocclusion rather than acts as a causative factor. Fig 30*A* shows a case complicated by the supernumerary tooth shown attached to the cast after its extraction. The x-ray picture shows its position to be labial to the permanent central incisor. No stretch of the imagination could hold this condition accountable for the constricted arches; yet it helped to

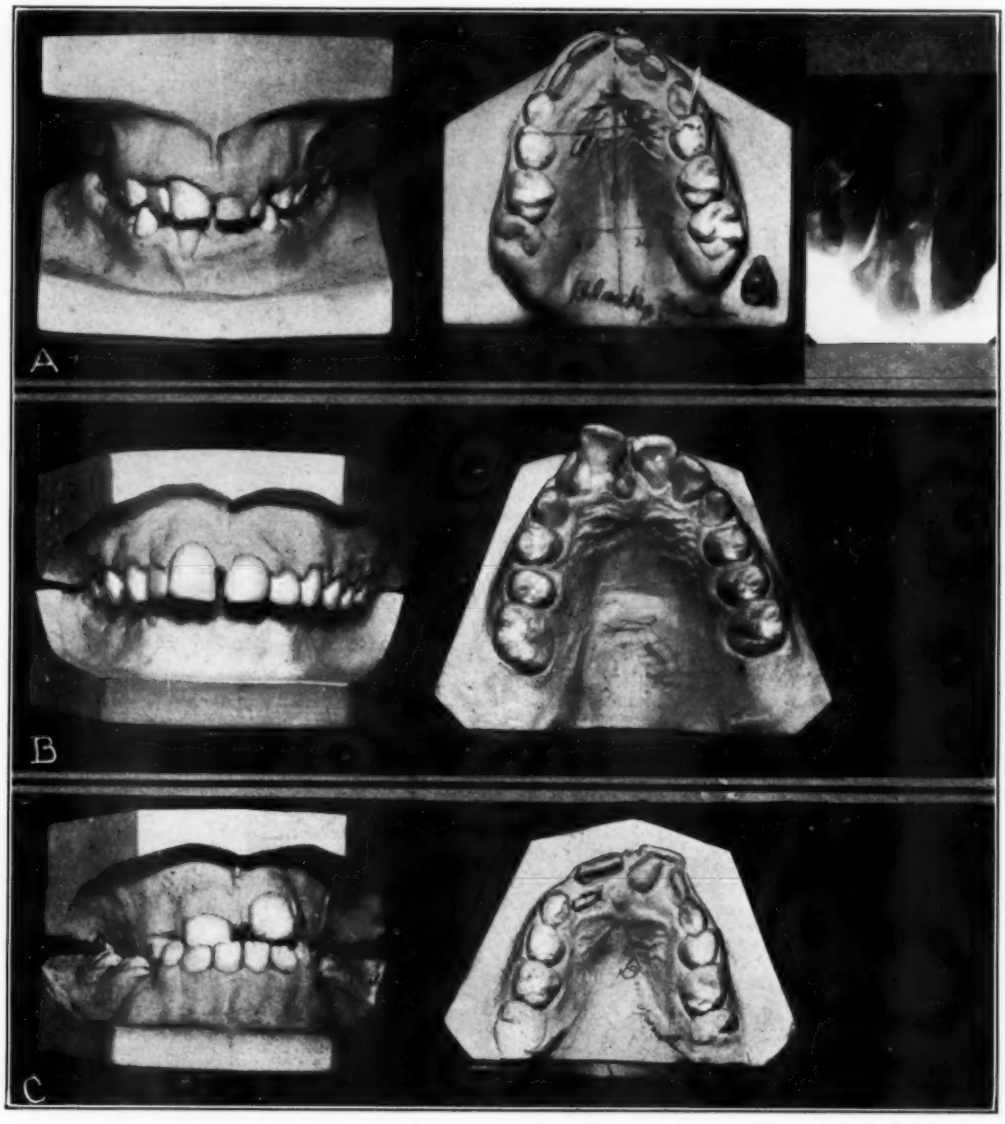


Fig. 30.—Existing malocclusions complicated by presence of supernumerary teeth.

complicate the development. Fig. 30*B* shows a different type malocclusion complicated by the irregularly shaped supernumerary tooth lying between the central incisors. Fig. 30*C* shows still a different type of this group of cases. The supernumerary tooth is almost invariably removed and treatment planned just as though the supernumerary had never existed.

It should be noted that this classification does not provide for those cases in which the presence of a supernumerary tooth has no apparent effect on a dentition. Fig. 31 shows a case which was treated and completed without consideration of the supernumerary incisor whose presence had no apparent effect on the irregularity. Its extraction, however, was advised in order to prevent possible injury to the central incisors.

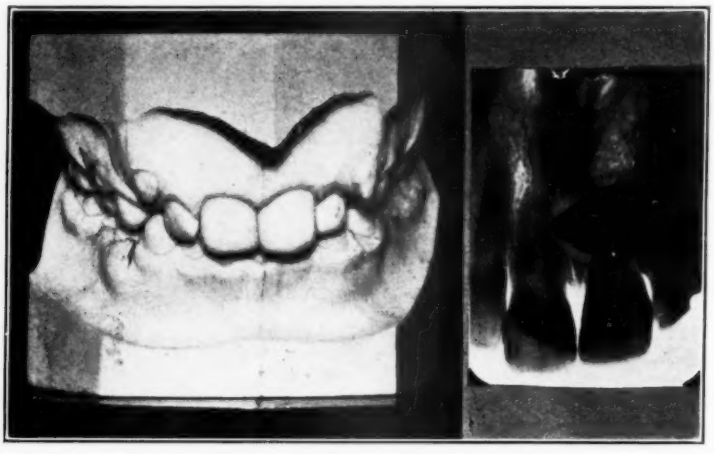


Fig. 31.—Supernumerary tooth which did not effect malocclusion.

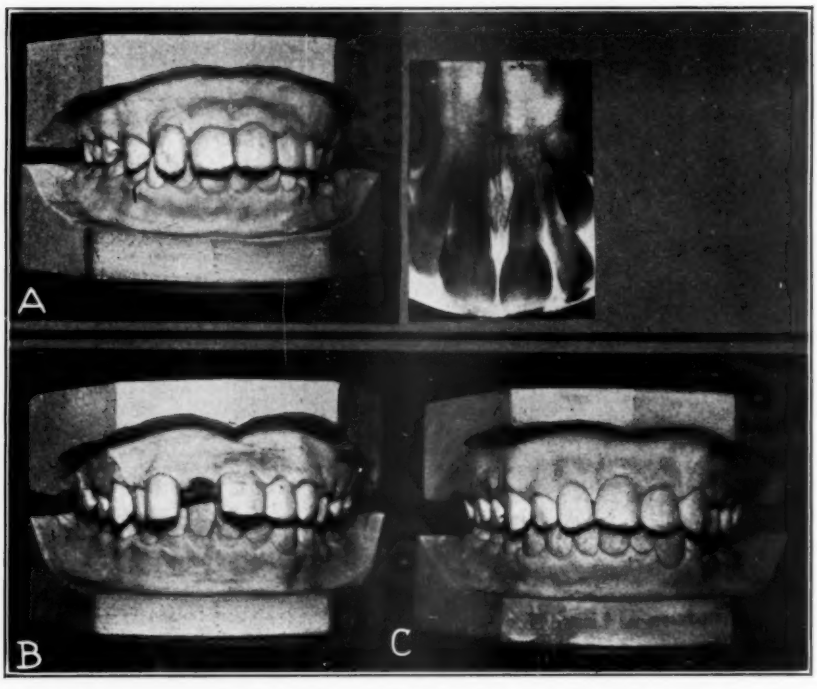


Fig. 32.—Supernumerary tooth as an aid in treatment: *A*, before treatment; *B*, case after loss of mandibular molar and maxillary central incisor; *C*, after treatment.

Occasionally supernumerary teeth are found that aid rather than complicate the treatment of a malocclusion. Such was the case of the supernumerary left lateral incisor shown in Fig. 32*A* which turned out to be a life-saver. Treatment of this case was started when the patient

was nine years of age. Several months afterward a mandibular first molar whose condition had been considered questionable abscessed, and necessitated removal. All appliances were removed at that time. The patient did not present himself at the office until two years later. In the meantime his maxillary right central incisor had become devitalized as a result of a baseball, thrown and missed. At this time the supernumerary tooth had erupted as shown in Fig. 32*B*. Treatment was then resumed with the object of eliminating bridgework by utilizing the supernumerary. This was accomplished with the end result as seen in Fig. 32*C*. Only trained observers now notice the discrepancy in the size of the right central incisor.

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8047 JENKINS ARCADE

ORTHODONTICS AS AN AID IN REPAIRING A CONGENITAL CLEFT

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IN presenting this report, I should like to preface these remarks with the explanation that it is a preliminary report. Additional stimulation of the maxillary bone is necessary to complete the process of eruption of a retained tooth.

Fig. 1.—Front and profile views before and after treatment.

History.—The patient, male, 12 years of age, of slight physical development, presented with a repaired double harelip and cleft palate, Type IV of Fitz-

Read at the annual meeting of the New York Society of Orthodontists, March 7, 1938.

gibbon's classification, where the cleft extends from the soft and hard palates through to the ridge on each side of the maxilla, causing a double harelip. At three months, he had been operated on for repair of the harelip, and at two years of age a second operation had been performed for the repair of the palatal cleft. He had been bottle fed after the operation, being unable to employ suction until repair of the lip, and his food had been carefully supervised subsequently with an earnest endeavor to maintain a balanced diet. (Fig. 1.)

Of the typical childhood diseases, he had had measles, chickenpox, and whooping cough. Tonsils and adenoids had been removed at seven years of age. At the time of the cleft palate operation, there was no attempt to repair the bony defect, and a large opening was left in the left maxilla.

Fig. 2.—Full mouth x-ray series showing congenital absence of maxillary second premolars and maxillary cleft.

Radiographic examination (Fig. 2) revealed in the left maxilla a palatal defect in the region of the premaxillary suture of a cleft 1½ cm. long and 1 cm. wide. Wedged in on either side of the border, approximating the lower circumference, were a retained permament lateral incisor and canine. The right and left maxillary second premolars were congenitally absent. All other permanent teeth were present up to and including the second molars, which were not erupted. In the mandible, all teeth were present up to and including the second molars, which were not yet erupted, and the buds of the third molars were seen just beginning to form.

Oral examination (Fig. 3) revealed a cross-bite with lingual version of the left maxillary central incisor and premolar, and the right maxillary second

deciduous molars of both sides were badly broken down, and there was general poor mouth hygiene. There was bunching of the mandibular anterior teeth with hypertrophic gingivae that bled at the lightest touch. The patient was a mouth-breather.

Fig. 3. Fig. 4.

Fig. 3.—Clinical picture showing cross-bite relation in the molar and incisor regions, before treatment.

Fig. 4.—After treatment, showing correction and recovery of lateral incisor.

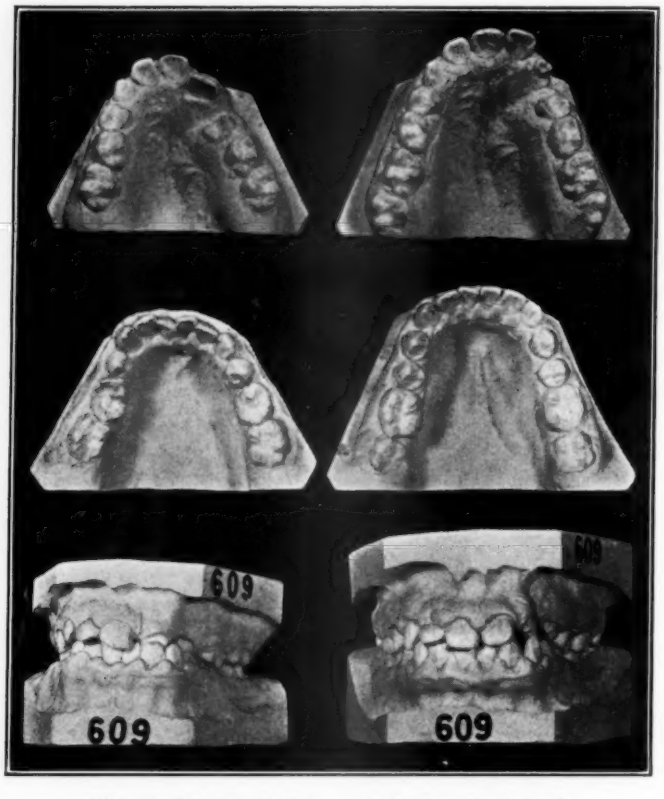

Fig. 5.—Models before and during treatment.

Attributed Etiology.—Early surgical interference had caused a disruption of the position of the maxillary tooth germs, and when the palate healed after the operation there was a contraction, causing a malposition of the left maxillary lateral incisor and canine, and contracture of the palatal width.

The loss of approximal contact through caries added further complications to disturb tooth conditions through drifting.

Diagnosis.—A mutilated neutroclusion resulting in a bilateral cross-bite of molar and premolar segments, and left maxillary incisors complicated by a congenital defect. There is general underdevelopment of the mandible with a crowding of the mandibular anterior teeth. (Fig. 5.)

Treatment Employed.—Bands with buccal tubes to accommodate 0.040 labial wire were cemented to the first permanent molars in the maxilla. These carried a soldered lingual arch that supported two auxiliary springs of 0.020 thickness, to move the left maxillary incisor labially, and the left premolar buccally (Fig. 6). The root end of the left maxillary central incisor was tipped distally and that of the premolar mesially, so as to create pressure on the surface of the border of the cleft, and stimulate through interstitial growth further development of the maxillary bone, which would help repair the congenital bony defect and thus offer aid in supporting the eruption of the retained lateral incisor and the canine. This was aided by a labial appliance. The central incisor and the first premolar were banded to permit more positive movement of these teeth as indicated.

Fig. 6.—Showing maxillary appliance in position.

Bilateral buccal expansion was introduced to correct the contracted arch,and the mandibular anterior teeth were realigned with a removable lingual arch that employed auxiliary springs for the forward and lateral stimulation.

Results Achieved.—This slow stimulation resulted in the eruption of the lateral incisor in the left maxilla and a downward eruption to the incisal plane of the left maxillary canine. The latter has still to achieve its place in the arch.

The palatal defect shows regeneration of the trabeculae and a filling in of this congenital hiatus. (Figs. 7 and 8.)

Prognosis.—The prognosis is quite favorable for the continued eruption and the final realization of the six anterior teeth in good alignment. There is reason to feel certain, by the reaction observed so far, that there will be a continued lessening of the congenital cleft through stimulation of root ends employed through natural function.

Observations and Conclusions.—In a case report before this society in 1936 by Dr. Earl Swinehart, a very interesting problem presented as a result of sur-

gical trauma in the removal of an ankylosed retained maxillary canine. Through surgical interference an excessive amount of bone tissue was sacrificed in its removal after the tooth did not yield to orthodontic persuasion. Dr. Swinehart attempted and did lessen this defect through orthodontics by stimulation of the alveolar structures with the agency of root pressure induced by an

Fig. 7.

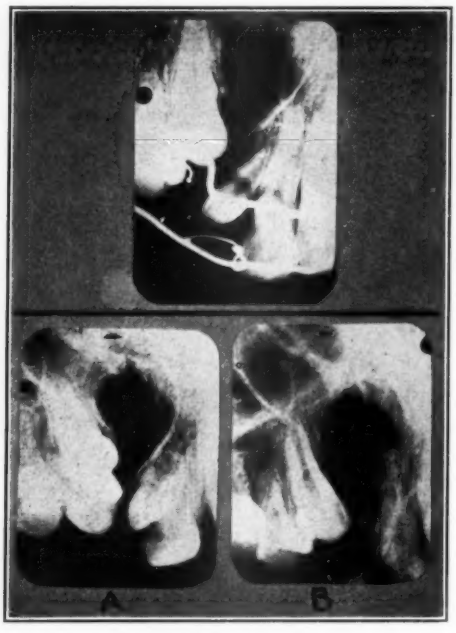

Fig. 8.

Fig. 7.—Intermediary stage of treatment, showing additional trabecular stimulation distal to the lateral incisor.

Fig. 8.—*A*, The palatal defect before interstitial stimulation. *B*, After canine and lateral had been brought down.

orthodontic appliance. This excellent presentation so closely paralleled the procedure that I had been employing for a year at that time, that I felt prompted to add this additional experience of bone stimulation within biologic limits that may suggest a new approach in considering treatment of a condition that confronts the orthodontist much more frequently in cases of congenital cleft palate, than the rarer ravages of surgical trauma.

THE ERUPTION OF AN IMPACTED AND ENCYSTED CANINE*

M. R. CHIPMAN, D.D.S., SPOKANE, WASH.

HISTORY.—Patient was 13 years of age at the time of presentation in October, 1934. Roentgenographic examination at this time revealed the presence of an unerupted maxillary left canine which was impacted and slightly lingual. There was also evidence of a retained deciduous canine, and a cystic formation accompanied by a large area of bone destruction. No history of pain or discomfort was revealed upon questioning.

A medical history revealed nothing except the usual run of childhood dietary upsets, the physician reporting the patient to be in perfect health.

The dental examination revealed neither cavities nor restorations of any kind. Further examination brought out the fact that the maxillary left lateral incisor and first premolars were of an extremely low vitality, with the remaining teeth reacting in a normal manner.

The exact etiology is unknown because of lack of earlier records.

Diagnosis.—Roentgenograms were taken in October, 1934, and the photographs and models were made of the case in April, 1935. Examination showed evidence of a Class I case with a mesial drifting of the maxillary left posterior segment. This drifting had been due to a premature loss of the maxillary left first deciduous molar approximately five years prior to examination in October, 1934. The roentgenographs revealed a dentigerous cyst involving the unerupted canine, lateral incisor, and first premolar, and the retained deciduous canine. Evidence of the beginning of lip and tongue biting was found, but this apparently had no connection with the case as I found it.

Treatment Employed.—The plans for treatment of this case at the time were to my mind both simple and direct; namely, removing the deciduous canine, eradicating the cystic area, establishing of normal occlusion of the maxillary left segment, placing a pin in the impacted canine, and bringing the tooth into normal position. However, the best laid plans sometimes go awry.

The original roentgenograms were taken on Oct. 3, 1934. The deciduous canine was removed the following day, and another roentgenogram taken on Nov. 8, 1934. We were not quite satisfied with conditions and decided to have Dr. John Williams, oral surgeon, open the area and remove any osseous structure which might interfere with our progress. This was done thirty days later, or on Dec. 8, 1934. Dr. Williams found this area filled with a thick yellowish fluid.

Because of the low vitality of the lateral incisor and first premolar, and the large area of bone destruction, curettement was kept to a minimum. On Jan. 15, 1935, another roentgenogram was taken which seemed to indicate that some slight change had taken place in the position of the tooth and in

*Read before the Thirty-sixth Annual Meeting of the American Association of Orthodontists, Los Angeles, Calif., July, 1938.

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Fig. 1.

the osseous structure. Roentgenograms were taken approximately six weeks apart until June 22, 1935, when it was decided that orthodontic interference was needed. Maxillary round labial and mandibular lingual arches were placed at this time, and with the aid of intermaxillary elastics the needed movement of the maxillary left segment was started. This movement was aided later by the use of a maxillary lingual arch with loop springs. In this manner occlusion was established and normal room provided for the maxillary left canine. Before the movement necessary to the establishment of occlusion, the movement of the canine seemed to have reached a standstill.

On April 10, 1936, a pin was placed and an attempt made to bring the canine into its normal position through the use of appliances. The attempt was rather futile, although a little advancement was made during the next few months as is evidenced by the roentgenograms from April 3 to Sept. 3, 1936. However, paternal interference with appliances was too much to overcome, and consequently I decided to let nature take its course, if any. All appliances, except the maxillary lingual arch, were removed, the lingual arch being used merely as a retainer in maintaining occlusion and the space needed for the canine. No further orthodontic effort was made, and the retainer was removed entirely on March 4, 1937. Roentgenograms were taken regularly until Oct. 8, 1937, the canine having attained occlusion and a normal position in the arch.

Fig. 14.

Results Achieved.—The last photograph taken on Dec. 31, 1937, shows the maxillary left canine in normal position with a fairly normal occlusion of the maxillary and mandibular dentures. Vitality gradually returned to the maxillary left lateral incisor and first premolar; tests revealed a full recovery in January, 1936. Tests applied between Dec. 8, 1934, the time the cyst was removed, and January, 1936, showed a steady increase in return to normal vitality. Check tests have been made several times during 1938 and all have proved to be normal.

Prognosis.—All examinations point to the fact that a favorable end result has been attained, and I can see no reason for difficulties in the future with this case.

Observation and Conclusion.—During the past years I have been pleasantly aware of the fact that I have had some considerable success with impacted canines, but after observing this case and several others recently, I am beginning to wonder if discretion is not better than valor. I was kept so busy repairing appliances and endeavoring to keep a pin in a place where it was not wanted that I was deluded with the idea that I was actually doing some orthodontics. Actual active orthodontic endeavor hardly covered a period of six months and that mainly was concerned with establishing occlusion and gaining space for the eruption of the cuspid.

As a result of this interesting case, I have, since 1936, been a little hesitant in applying orthodontic measures to impacted canines unless the case actually demands interference due to the unfavorable tooth position. My policy has been concerned more with establishing occlusion, maintaining space for the unerupted tooth, removing obstructions in the way of overlying bone, and watchful waiting until such a time as other means seem advisable. I believe it has paid good dividends.

The most interesting feature of the case is not orthodontic procedure, but the manner in which the osseous structure has followed the eruption of the cuspid, and the apparently normal manner in which bone formation has taken place around the lateral incisor and first premolar. Dr. Williams, after removing the cyst, seemed quite convinced that the root of the lateral incisor was apparently denuded of bone on the distal portion.

This series of roentgenograms is tendered to the American Association of Orthodontists not only as a record of an impacted cuspid, but also for any value you may place on it as a record of the eruption of a tooth with the accompanying osseous changes.

1251-2 Paulsen Medical and Dental Bldg.

CASE OF IMPACTED FIRST PERMANENT MOLARS

MABEL F. THOMAS, D.D.S., NEWARK, N. J.

THE case which I have selected to report is that of a boy seven years of age. The first permanent molars were impacted, and in erupting caused the premature loss of the deciduous second molars. The maxillary left permanent lateral incisor was missing, and on the right side only the crown of the tooth was present, with no further development apparent.

The patient was first seen in March, 1936, and was of average height and with rather slight framework. So far as can be determined, he was a perfectly healthy, normal child; he ate well and was never ill, with the exception of the usual childhood diseases of mumps, measles, and chickenpox. His diet was very carefully supervised; in other words, he had been reared according to the pediatrician's advice, with the common fault of having food too soft and not hard enough substances to chew. He had never sucked his thumb or fingers, and with the exception of a lisp (a hangover from the days when he was first learning to talk), I could find no habits which would have any bearing upon the development of his teeth and jaws. The lisp, a disturbance in lip and tongue function, was an acquired habit, and is being corrected now only by constant care and special attention to the pronunciation of words.

At the beginning of treatment, he had present in his mouth, in addition to the normal deciduous teeth, three mandibular permanent incisors, with little or no room for the fourth, and the mandibular permanent first molars. In the maxilla, the two permanent central incisors were erupting with mesial incisal edges tipped toward the gingiva. The first molars were impacted with the mesial occlusal margin locked in under the distal surface of the deciduous second molars; the distal root of the deciduous tooth had been absorbed and very little of the approximal surface remained.

Fig. 1 shows the photograph of patient at the beginning of treatment. Fig. 2 shows models made at the beginning of treatment and about a year and a half later. The maxillary deciduous lateral incisors were small well-formed teeth with no sign of loosening. X-ray examination showed that the left lateral incisor had no successor, but on the right side in the lateral region there was a crypt containing a peg-shaped crown of a tooth. Watching this tooth, radiographically, it appears to have grown slightly longer in the two years that the patient has been under observation (Fig. 3). Fig. 4 shows x-ray pictures of the molar and bicuspid areas, showing impacted maxillary permanent molar and resorption of the deciduous second molar root. Fig. 5 shows pictures of the same areas, taken three months later, showing premature loss of the deciduous tooth and improvement in the inclination of the permanent tooth.

The first step in the treatment of the case consisted in placing separations between the maxillary permanent first molars and the deciduous second molars in an effort to direct the developing permanent molars straight downward in-

Presented before the New York Society of Orthodontists, New York City, March 7, 1938.

stead of obliquely as they were erupting. The separations were tightened and increased in thickness from time to time, until the mesial occlusal edge of the erupting tooth was freed from the deciduous molar. This treatment covered a period of about three months, and at the end of that period the inclination of the molars was greatly improved. When the patient was 7½ years of age, or

Fig. 1.

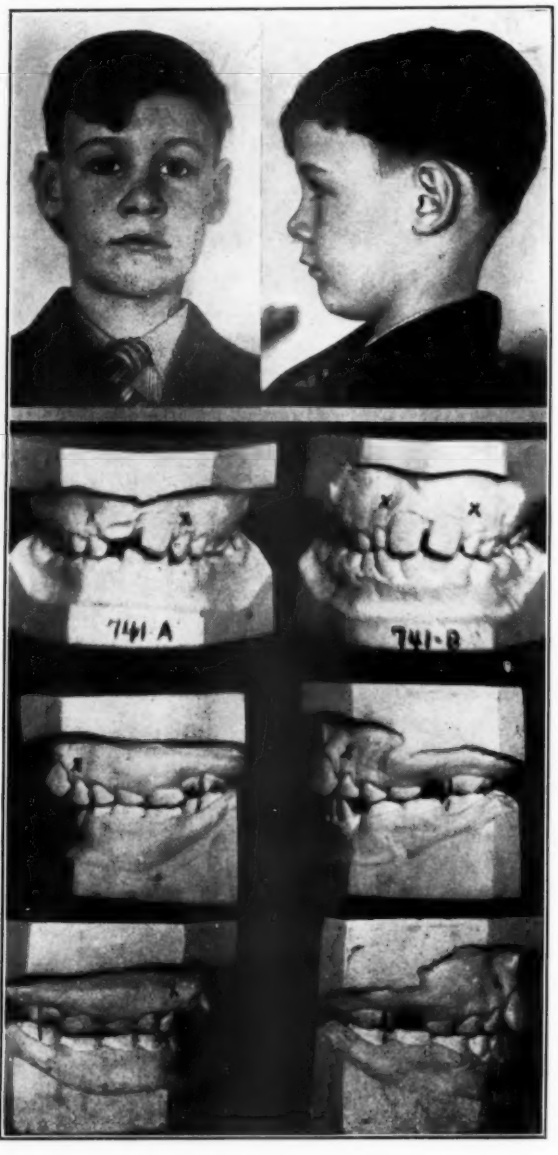

Fig. 2.

just about three months after treatment was begun, the maxillary deciduous second molars were lost, complete resorption of the roots having taken place.

A mandibular appliance had already been placed in the mouth, and the arch was being expanded to make room for the mandibular left lateral incisor, which was in lingual position.

When the maxillary permanent molars had erupted sufficiently, they were banded and a labial arch was built. The mandibular appliance was changed to a

labial arch, with lingual wires soldered to molar bands extending to canines for lateral expansion. Intermaxillary elastics were worn. In the mandible, the left deciduous canine was prematurely lost, as it usually is when the lateral incisor erupts lingually and is moved labially into alignment. After about nine months of this treatment, normal occlusion was established, and all appliances were

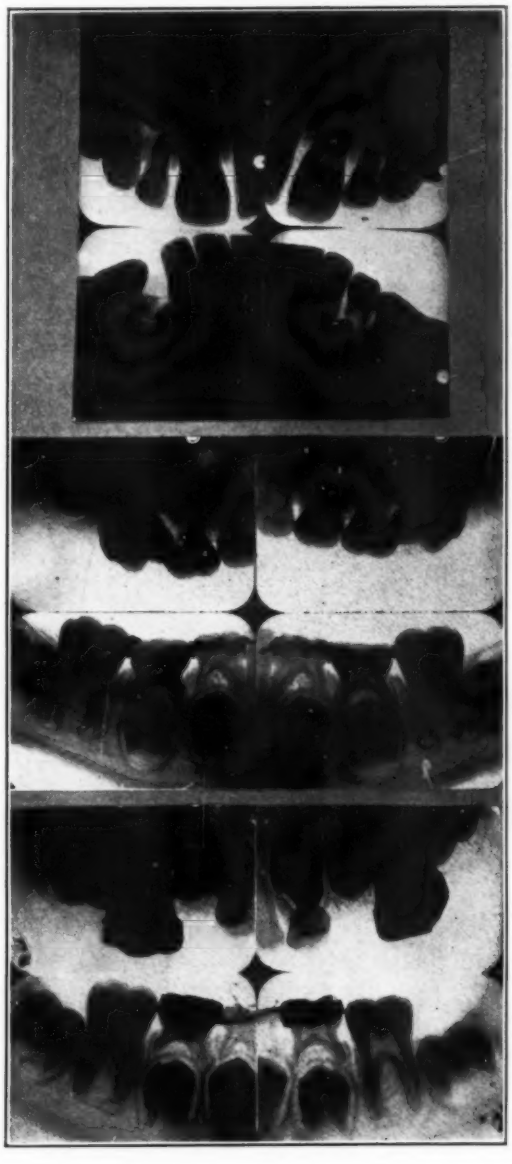

Fig. 3.

Fig. 4.

Fig. 5.

removed. A retainer was made for the mandible, which was worn for a period of about four months, at which time it was removed and the case kept under observation.

Upon examination at the present time, I find that the spaces in the maxillary second bicuspid area have closed slightly, and so has the mandibular left canine area. I believe that we shall have to reset the arches and institute further treatment when the premolars are ready to erupt.

CASE REPORT OF INTERNAL RESORPTION

MEYER EGGNATZ, D.D.S., BALTIMORE, MD.

THIS is a true case of internal resorption of dentine, resulting in "pink spots" as first described by Mummery.

This patient has a history of severe trauma on two occasions. The periodontal fibers were torn, and in order to have reattachment (functional repair) that part of the cementum from which the fibers were torn is absorbed. Usually the resorption process ceases, and new secondary cementum is built in, and as it builds in, new periodontal fibers are attached.

In this case because of repeated severe trauma, the resorption process did not cease and lateral perforations from the mesial and distal sides resulted. When the pulp was exposed, the embryonic connective tissue was changed to adult connective tissue. Adult connective tissue is incapable of producing dentine. It can produce only cementum or bone. In this case the embryonic pulp tissue was converted into adult connective tissue. Because a wide space was created by the perforation, a new alveolar process was built, in a horizontal direction. If this condition had remained in the mouth longer—complete repair —that is, a new alveolar process, periodontal fibers and secondary cementum on the exposed dentinal walls would result. In many other areas of the pulp, developing bone of the intramembranous type is seen.

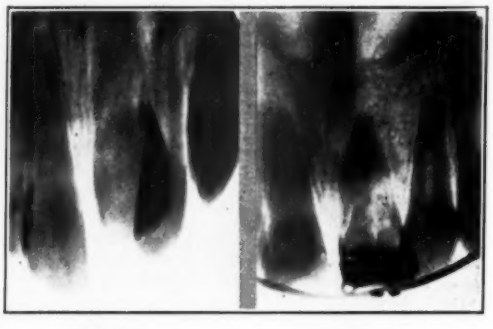

Fig. 1. Fig. 2.

This specimen verifies the explanation given by M. S. Aisenberg in a paper on "Alveolar Bone in the Pulp Chamber." He states, "When the embryonic pulp is exposed by fractures and resorption, the adult connective tissue of the periodontal membrane invades and changes the pulp to adult connective tissue. Embryonic tissue is necessary for dentine formation. Adult connective tissue can produce only cementum and bone. Where the resorption space is wide, a new alveolar process with new peridontal fibers attached to secondary cementum deposited on the dentinal walls will result."

Read before the Washington-Baltimore Society of Orthodontists, April 21, 1938, at Baltimore.

Figs. 1 and 2 show x-ray pictures before and after treatment. In Fig. 1 we see two resorption areas: one on the mesial, the other on the distal side. This was overlooked, as we sometimes see these areas on normal teeth. Fig 2 shows internal resorption, just prior to extraction.

Fig. 3 is a photograph of the mesial side showing the wide perforation. Adult connective tissue invaded the pulp changing the pulp tissue which is embryonic to adult connective tissue. Because the space was wide, and because pressure or pull was exerted on the tooth, a new alveolar process grew in from the alveolar bone of the tooth socket. There was not enough time for the connective tissue to become organized as a periodontal membrane. Sometimes this alveolar bone becomes ankylosed to the dentinal wall.

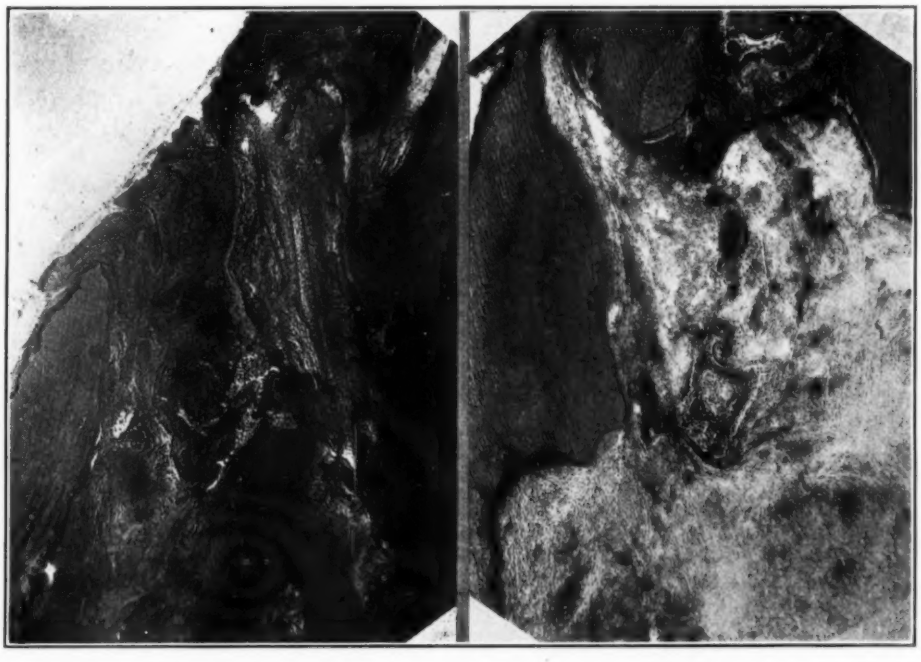

Fig. 3. Fig. 4.

To prove the statement that adult connective tissue can produce only bone or cementum, we show in Fig. 4 another area in the pulp. The embryonic tissue has been changed to adult type, and we see an area of bone developing, intramembranous type.

REFERENCE

Aisenberg, M. S.: Alveolar Bone in the Pulp Chamber, J. Dent. Research, 1938.

ORTHODONTIC TREATMENT OF A FRACTURED MANDIBLE COMPLICATED BY OSTEOMYELITIS

KYRLE W. PREIS, D.D.S., BALTIMORE, MD.

A STEADY diet of fractures is not particularly desirable in an orthodontic practice. There are times, however, when the orthodontist, with his specialized technique of band and arch construction, can be a most welcome adjunct in the treatment of complicated and prolonged fracture cases. The preservation of arch form and the best possible occlusion of teeth are his goal.

Fortunately, a great many fractures of the maxilla and the mandible are simple, without infection; indeed, often with the mucous membrane unbroken. These fractures quickly respond to simple wiring treatment, and in general the outcome is favorable. All fractures, however, are not so easily disposed of, and it is of one such case that I now wish to report.

Fig. 1.

Fig. 2.

Figs. 1 and 2.—August, 1933.

On July 9, 1933, the patient, a young man, fell asleep while driving, struck a telegraph pole, and awoke in a New England Hospital, with a fracture in the symphysis of the mandible. For almost three weeks he remained in the hospital with his mandibular teeth wired to the maxillary teeth. As the patient's health

Read before the Washington-Baltimore Society of Orthodontists, April 21, 1938, at Baltimore.

rapidly declined, and as it also became clearly evident a bad osteomyelitis infection had set in, the boy's parents became uneasy and decided to bring him home to Baltimore.

Fig. 3.

Fig. 4.

Fig. 5.

Fig. 3.—September, 1933.
Fig. 4.—October, 1933.
Fig. 5.—November, 1933.

My first contact with the patient was on July 27, at the Union Memorial Hospital. His jaws were still wired, with swelling and extreme soreness in the left condyloid region. This condition may have been due to prolonged muscular ten-

Fig. 6.

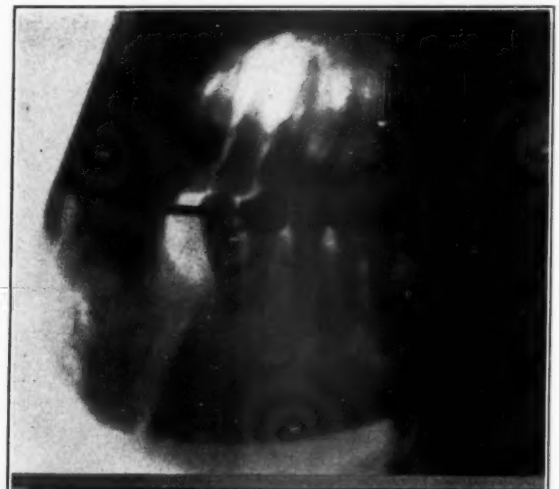

Fig. 7.

Fig. 8.

Fig. 6.—March, 1934.
Fig. 7.—July, 1934.
Fig. 8.—May, 1935.

sion or traumatic injury in the glenoid fossa. The exudate at the symphysis was excessive and nauseating, the temperature 103°. That evening wires were removed, impressions taken, separation was inserted between the first and second bicuspids and between the first and second permanent molars.

The splint was first constructed on a model, on which the first permanent molars and first bicuspids were carefully carved. The bicuspid and molar bands were made of platinous gold; the heavy buccal tubing, and the square arch wire of noncorrosive metal. The bands were made a trifle larger than usual, requiring but a minimum of pressure to seat.

On the buccal surfaces of the molar bands was soldered very heavy rectangular tubing, permitting the insertion of a square labial arch.

On the buccal surfaces of the bicuspid bands was soldered U-fashioned soft ribbon arch wire, with the ends extending long enough to wrap securely around the labial arch, which was carefully adjusted on the model.

A small hook, which served as a tie spot for a ligature preventing the arch from sliding, was attached on both sides just forward of its insertion into the buccal tubing.

On July 29, the four bands were individually cemented; the arch was placed in position while the patient was under a general anesthetic, the operation being too painful to attempt otherwise.

At that time the teeth were sensitive and gums inflamed, and there was free pus draining into his mouth, there being no external opening. That evening the patient seemed to be resting fairly comfortably. After two more days he was dismissed from the hospital.

On August 1, the patient came to the office, with pus settling directly under the fracture. When we attempted to flush the fracture with Dakin's solution, great pain was experienced because of the increasing tension already present.

On August 2, arrangements were made to have the abscess opened at Union Memorial Hospital; after a jostling cab ride, the patient arrived with the abscess already draining. We enlarged the opening, however, and a rubber drain was inserted. The relieving of the pressure added greatly to the patient's comfort.

From August to December the patient was seen at short intervals, at which time x-ray pictures were taken. Many particles of bone were removed through both the oral and the submental openings. The irrigations aided in subduing the odor, and I believe accelerated the exfoliation of necrotic bone. Treatment was followed along these lines until the appliance was removed in June of the following year.

Throughout treatment, the diet included generous amounts of milk, cod-liver oil, green vegetables, and calcium phosphate in tablet form.

Particularly significant from an orthodontic viewpoint is the fact that the proper occlusion of the teeth was maintained by the use of a rather rigid splint. This type of appliance permitted free movement of the mandible, normal feeding, a greater degree of cleanliness, and ideal fixation, during a period when muscular stress or other factors might easily have caused either median or lateral displacement of the mandible with a corresponding distorted facial appearance.

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CELLULITIS

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THE terms cellulitis, phlegmon, adenitis, osteitis, osteomyelitis, and necrosis are confusing to many. The words are used by some almost synonymously, but when accurately understood, they signify definite processes or stages in the response of soft or hard tissues to infection.

Cellulitis is a diffused or circumscribed inflammation of cellular or connective tissues, as a result of bacterial infection. This type of infection is often found in the neck, cheek, lips, or any of the soft tissues of the face and oral cavity. The process of inflammation may or may not extend to the stage of suppuration or abscess formation. It may be acute or chronic.

When bone is inflamed because of bacterial infection, the blood supply is increased. This is nature's first protective measure. Leucocyte infiltration is also stimulated, and, if the inflammation is acute, suppuration will invariably follow. The bone, however, is protected by a pyogenic membrane, and therefore the infection may be localized. This is called *suppurative (acute) osteitis.* The teeth become tender and elongated. The general symptoms may be severe with intense pain, chills, and fever. The adjacent cellular tissues become infiltrated with the organisms, resulting in cellulitis. On the other hand, if the inflammation is chronic, nature's response is sclerosis, or an increased density of the bone. This is called *condensing osteitis.*

If the inflammation process extends to the central core of cancellous bone in the maxilla or in the mandible (these bones do not have a bone marrow), the infection and its effects may be so overwhelming that the Haversian canals and foramina, the circulatory pathways, become plugged by bacterial thrombi which shut off the circulation and produce death of the adjacent bone *en masse.* This process is called *osteomyelitis.* It may be localized or may involve the entire bone, terminating in *necrosis* with the formation of sequestra.

The process then is a focus of infection, extending itself to the soft or cellular tissues, developing cellulitis which may or may not result in suppuration for reasons we will discuss later and which may or may not develop into osteomyelitis and subsequent necrosis.

Cellulitis may exist without osteomyelitis or necrosis. It is commonly a result of acute osteitis and is frequently associated with *adenitis* or lymph gland infection. Carious teeth with infected pulps, pericemental disease, pericoronal infection about incompletely erupted teeth are common etiological factors. Cellulitis may originate from sources other than teeth or bone. Furuncles, boils, pimples which have been squeezed, hematogenous infections, syphilis, fractures, and metallic poisons are frequent sources of infection leading to cellulitis.

Cellulitis may occur at any age, and it may or may not break down and suppurate. The formation of pus depends greatly on the type of organism, the virulence of the organism, and the physical resistance of the patient.

TYPE OF ORGANISM

The types of organisms found in cellulitis are 1. *Staphylococcus aureus* and *Staphylococcus albus* and 2. streptococcus, including the *Streptococcus hemolyticus.*

The *Staphylococcus aureus* has a high virulence. It spreads by the blood rather than by the lymph vessels and may cause multiple abscesses in the kidney, heart, and other organs. It is found in carbuncles and boils and causes an intense local reaction. It may attack any tissue, but each lesion will be sharply defined. This is due to an effect that this organism has on the neighboring lymph channels; it plugs them with fibrin and thereby assists localization and prevents spreading of the infection through the lymph channels. The *Staphylococcus aureus* acts on tissue fats and is greatly responsible for the rancid odor of suppuration. One can be reasonably certain that when markedly thick pus appears, it is probably due to staphylococcus infection. The less severe types of cellulitis are probably due to the staphylococci; and virulence of the organism and the resistance of the patient determine the extent and seriousness of the process.

The streptococcus unlike the staphylococcus does not localize but is associated with diffused or spreading infections, with violent inflammation and systemic reactions. It travels rapidly through the interstitial tissues along the planes of cleavage. The toxins are frequently absorbed through the lymphatics and are the cause of marked swelling of the regional lymph nodes. The hemolytic type of streptococcus breaks down the red cells, liberating the hemoglobin and producing a hypochromic or secondary anemia.

POSTOPERATIVE CELLULITIS

The etiology in some cases is frequently puzzling. I am not including in this discussion edematous swellings due to trauma. Several other factors, however, may have been overlooked, even though the rules of asepsis may have been strictly followed and the surgical trauma induced may not have been excessive.

1. The local circulation may have been poor. Condensing osteitis restricts the blood supply and lowers the local resistance of bone to infection. Novocain with a marked vasoconstricting action will also lower the local resistance of tissues by restricting the circulation for too long a time.

2. The general circulation may have been impaired. Arteriosclerosis favors infection by reducing the nutrition of the tissues and lessening their resistance to infection.

3. Primary or secondary anemia, other blood dyscrasias, uncontrolled diabetes, low vitamin C blood level, marked cardiac disease, and even fatigue can predispose any of our simple operative cases to cellulitis.

4. Operating on tissue which is acutely infected requires a surgeon's best judgment. Rarely, in these cases, is a surgeon's first act carried beyond incision for drainage. Unintelligent treatment of pericoronal infections around third molars has caused many despairing moments for dentists and their patients.

Symptoms.—Acute infections cannot be overcome without inflammation. Inflammation is nature's way of fighting infections. What happens when nature responds to an acute infection? General effects occur, such as chills. Chills, probably caused by a momentary spasm of the peripheral blood vessels, may precede an immediate elevation of body temperature. This is nature's first step. Fever stimulates all chemical and cellular activities, increasing tissue and body resistance. The exact mechanism of fever is not clearly understood, although it is attributed to a stimulation of the oxidative processes of the body by toxins in the blood. Nature also increases the pulse rate in an effort to supply the tissues more readily with oxidized blood and to carry off waste products more expeditiously.

Fig. 1.

In all cases of continued high temperature, however, the oxidation of the tissues is largely increased. There is an increase in urea, the product of oxidation of nitrogenous tissues (muscles and glands); the nutritive processes are profoundly disturbed; and the essential elements of the tissues undergo degenerative changes. The eyes become injected, the bowels constipated, the urine scanty, and the tissues markedly dehydrated.

A rapid change takes place in the differential count of the white blood cells. The leucocytes are filtered profusely through the vessel walls to surround the invading bacteria as nature's first line of defense. Blood serum finds its way into the surrounding tissues, and a contest is started between the defensive mechanism of the entire body and the bacteria.

Locally, an intense process is taking place. If the focus is a tooth or within bone, severe pain may precede soft tissue swelling, owing to the confinement of inflammation within bony walls. When the infection involves the

soft tissues, cellulitis develops, and the tissues become hard, reddened, and glossy. The swelling is tender and hot. If the muscles of mastication are involved, trismus results. The tongue becomes dry from fever dehydration. The adjoining lymphatic vessels are tender. The extent of symptoms is directly proportional to the amount of tissue involved and to the severity of toxin absorption, and is inversely proportional to the resistance of the patient.

Deep-seated infections which burrow along fascial planes are more severe because they are usually due to streptococcus and do not localize readily. The toxemia is greater, and the burden on the patient's resistance is seriously increased.

When the swelling has become hard, the extreme pressure caused by the inflammatory exudate and increased number of blood cells lessens the active circulation and further lowers the tissue resistance. If it extends to the neck or throat, difficulty in swallowing or even respiratory embarrassment may be present.

A small amount of pus may form by cell liquefaction without a complete breakdown of the tissues and extreme suppuration. A barrier of leucocytes formed around the focus limits the infection, and, if the resisting powers are strong enough, further pus formation may be checked, the fluid absorbed by the lymphatics, and the disintegrated cells removed by phagocytes. If, however, the predominating bacteria are staphylococci, a large abscess will form and will seek the path of least resistance for drainage.

TREATMENT

We have learned the following: Nature provides a defense against this process by increasing the body temperature and by increasing the pulse rate. Both of these processes, however, increase tissue oxidation and promote dehydration. Conservation of the patient's resistance is imperative, because of the tremendous tax placed on the body's defensive mechanism. When the tissues have suppurated, the pus will tend to seek the path of least resistance for drainage.

These are fundamental factors, and our treatment should assist these processes and not oppose them. Increased body temperature is nature's method of removing and killing injurious agents and of repairing injury. This suggests the important therapeutic use of heat. Heat applied to the outside of the face or neck and hot irrigations used inside the mouth support nature's effort to combat infection; however, when we are treating an acute cellulitis of the face, mouth, or neck, heat should be used carefully. Heat hastens the formation of pus or assists resolution by increasing tissue resistance; it encourages localization of pus when such a process is inevitable; it causes the swelling to enlarge by increasing the circulation and tissue fluids; and it hastens "pointing" when it is unavoidable.

If an abscess is forming external to fascial or muscle planes, the abscess will point extraorally, and, if internal to these planes, it will seek the path of least resistance and gain intraoral vent.

Technique of Flaxseed Poulticing.—Moist heat is more soothing and more penetrating than dry heat, but unless used correctly, it is of little therapeutic

value. When heat is applied to the outside of the face, as in the use of flaxseed poultices, the temperature should be maintained for at least one hour to obtain sufficient penetration. This may require a renewal of the poulticing medium three or more times during the hour. It accomplishes little to have a cold or lukewarm poultice in contact with the affected parts. The pack should be large enough to cover more than the affected tissues, and it should be sufficiently thick to retain the heat. The size and thickness of a hand is usually adequate.

Supportive Treatment.—Fluids are forced to replace those lost during fever, to dilute the waste products, and to expedite their removal. The general ability to assimilate food has been lessened, and, therefore, the patient is given easily digested foods in small quantities and at frequent intervals. If the infection has been prolonged, the vitamin C blood level is lowered, and sixteen to twenty-four ounces of citrus fruits or tomato juices should be included in the diet. Elimination should be watched.

Since rest is nature's main method of repair, the patient should be hospitalized or else put to bed at home with the proper attendance. Transportation of a patient with an elevated temperature to and from dental offices is debilitating, and might, because of the increased exhaustion, change the entire aspect of the case.

Loss of sleep because of pain is weakening. Aspirin, anacin, pyramidon, and other anodynes may be effective. Morphine, codeine, dilaudid, or other opiates may have to be used. Nembutal, phenobarbital, and other barbiturates are used as hypnotics to produce sleep primarily, but sometimes to relieve pain secondarily. If the infection is long-standing—two or three weeks—the red blood cells are affected and they become hypochromic. Iron therapy is popularly used to increase the hemoglobin in such cases.

The *Streptococcus hemolyticus* has a very harmful effect on the red blood cells, causing their hemolysis or dissolution and breakdown. This organism also affects the regenerative properties of the bone marrow, the producing of red cells. The hemolytic streptococcic infections have a high mortality rate depending on the age of the patient, location and extent of the local lesion, the presence of debilitating diseases, the presence or absence of bacteremia, and possibly the type of hemolytic streptococci involved. Dr. Keefer, of Harvard, reports that the highest fatality rate is observed in patients under five or over fifty; when bacteremia is present, the fatality rate is in the neighborhood of 70 per cent, and it is highest in patients with cellulitis and erysipelas. He further reports that sulfanilamide is effective in delaying the death of mice infected with many lethal doses of hemolytic streptococci. In man the most suggestive evidence that this drug has a beneficial effect is seen in the cases of hemolytic streptococcic meningitis with recovery following its use, and in the cases of hemolytic streptococcic bacteremia which it has favorably influenced.

Once sulfanilamide is given, its administration should be continued until recovery, although the dose may vary because of the demands and vicissitudes of the case. The temperature drops and the swelling decreases, suggesting

that the patient is well on the road to recovery. But the antibodies do not seem to be stimulated further, and, if treatment is discontinued, a second innoculation may prove fatal. If a hemolytic streptococcus can be demonstrated as the predominant organism, sulfanilamide should be used. Blood studies should be made daily, and the patient watched carefully.

Incision for Drainage.—The time elected for incision for drainage varies. Usually it lies somewhere between the fourth and the eighth days after the first evidence of fever. There may be a definite pointing or softening of the surface tissues when fluctuation is evident. When two fingers are alternately pressed on either side of the swelling, a sense of the presence of fluid is gained. It may be difficult to obtain fluctuation because of the depth of the abscess and excessive infiltration of the tissues. A differential white blood count may be an aid in choosing the operating time.

Fig. 2. Fig. 3.

The first surgery is rarely more than incision for drainage. The physical principle of having the outlet at the lowest level in order to drain a cavity properly is involved. Otherwise improper location of the incision will allow the infection to burrow and necessitate additional incisions. Incisions should also be long enough to assist drainage; the surgeon should realize at the time of operating that the opening will tend to heal and unless it is made sufficiently extensive, the drainage may not be complete before the incision has closed.

If the abscess is deep, incise through the skin only, and with a pair of hemostats bluntly dissect the deeper tissues by forcing the end of the instrument through the overlying tissues and diverging the beaks or ends. The size of the hemostat may be graded from small to large until finally the gloved finger may be used to dilate the tissues further. A cigarette drain made with rubber dam or a fenestrated rubber tubing sutured in place may be preferred. More than one incision may be necessary in extensive abscesses or separate abscess formation, when through and through drainage is obtained by passing the drain from the first opening through the second incision, or vice versa.

Anesthesia.—Choice of anesthetic in the surgical care of cellulitis is important. The danger of inhalation of purulent matter during general anesthesia when the pharyngeal and laryngeal reflexes are absent is significant. If the abscess is draining intraorally or if intraoral drainage is to be obtained, extreme care must be practiced in the administration of a general anesthesia for fear of bronchial or pulmonary infections which might follow direct inhalations of pus. Pharyngeal swelling may restrict the respiratory passages sufficiently to make a general inhalation anesthetic impractical. In such instances, premedication of 1½ gr. nembutal one hour before operation, supported further in some cases by $\frac{1}{100}$ gr. of scopolamine or $\frac{1}{6}$ gr. of morphine will assist in the administration of local anesthesia to follow.

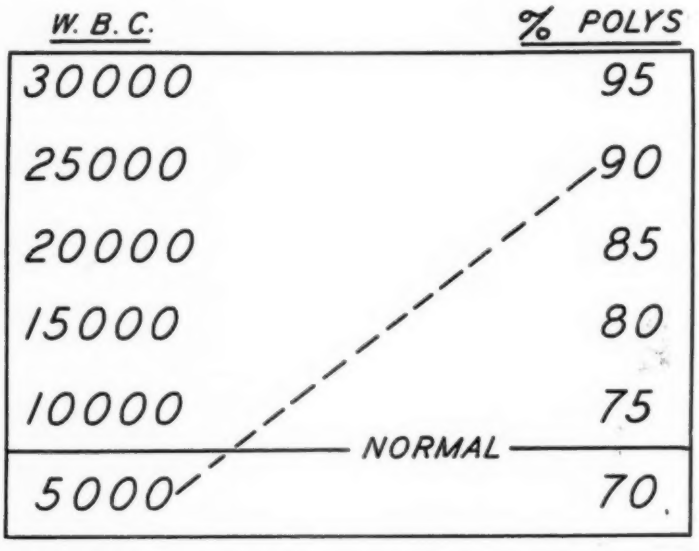

Chart 1.—Fatal cases show a rising line as above. Horizontal lines suggest a more favorable prognosis. If the polymorphonuclears are 85 to 90 per cent of the white blood cells, it is usually time to incise for drainage.

The fundamental principle of not using local anesthesia in acutely inflamed tissues might seem contradictory. There is no doubt that general anesthesia is the choice if the danger of pulmonary complications is not imminent. With this as a positive threat, however, the careful use of novocain is preferable. The tissues superficial to the abscess cavity are slowly infiltrated with novocain solution in order to obtain insensitivity of the area to be incised, and the operation of incision for drainage proceeds as previously described. Please note that the solution is discharged from the syringe slowly and without undue pressure. Also, I need merely mention that the face or area is to be properly cleansed with soap and water and painted with a 3 per cent or weaker solution of iodine, followed by alcohol.

After drainage, the temperature will drop within a few hours, and the severe pain caused by pressure is almost immediately relieved. The success of an operation is not measured by the brilliancy of operating. The successful recovery of patients is measured by the general restitution to good health and position of usefulness. A good dentist must cope with unexpected difficulties which occur suddenly. The following are additional factors helpful in the

care of the patient with cellulitis: (1) proper nourishment to hasten recovery; (2) rest and warmth; (3) maintenance of drainage until all suppuration has ceased. (Not until a final physical examination is made should the patient be allowed to "go back to the job.") (4) Finally, if the lesion was of dental origin, removal of the teeth with the necessary precautions, but not until after the acute symptoms have subsided.

Throughout the entire process, the treatment should be fundamentally sound; the surgeon should realize at all times that the patient is combating not only a marked local disturbance but also a general body reaction which may be attended by local deformities, by physical impairment, and possibly by death.

29 Commonwealth Avenue

THE PRODUCTION OF DENTAL CARIES IN ALBINO RATS BY THE INCLUSION OF MICROFRACTIONS OF ARSENIC IN THEIR DIET

A Preliminary Report

James D. Grace, D.D.S., Ann Arbor, Mich.

IN 1931 Hoppert, Webber, and Canniff[1] announced that dental caries resulted in albino rats fed that portion of ground corn meal which failed to pass through a 60 mesh sieve. Subsequently Klein and McCollum[2] attributed such caries to a phosphorus deficiency of the caries-producing diet. In 1934 C. A. Lilly and Leona Wiley[3] repeated the above experiment successfully and in addition performed phosphorus and calcium studies, concluding that the caries was not due to the inability of the animal to maintain a calcium-phosphorus balance, "since the greatest retention of these two elements was found in the rats which showed the greatest incidence of caries." In addition, these two investigators fed the finely divided corn meal which passed through the 60 mesh sieve, but were unsuccessful in producing carious lesions. They also fed another group of rats on the coarse diet, after cooking it for two and one-half hours, and this group also was negative for caries. Their conclusions were that "the incidence of caries was related to the physical form of the food (corn meal) and not to the calcium or phosphorus values of the diet."

It is difficult to accept the theory that the particle size of a diet, on account of a wedging action, could result in fracture of a tooth, but a relatively large particle could produce impaction in an occlusal pit or in a sulcus, permitting an unknown factor to exert a destructive action on the enamel, and, when this action had undermined the crown of the tooth, masticating stress on a hard particle could crush the crown. In everyday dental practice similar cases are observed. In this connection it might be mentioned that a sound, normal tooth will withstand relatively tremendous pressure. This fact is, of course, well understood by members of the dental profession.

Following out my hypothesis that corn kernels contained an unknown substance of powerful disintegrating ability, arsenic, on account of its wide distribution in soils, was decided upon as the logical subject for investigation. Therefore a sample of corn meal was analyzed for arsenic (Gutzeit method), and the result was positive. This determination was purely qualitative. A survey of the literature on this subject disclosed a value of 0.3 mg. per kilogram reported by Jadin and Astruc,[4] and one of 0.0001 per cent by the Kentucky Agricultural Experiment Station.[5] This latter value would indicate approximately 1 mg. of arsenic per kilogram. In this connection it is interesting to conjecture the possible variations in analyses from different sections of the country.

The next step was to analyze carious human teeth. The debris from twelve carious human teeth was curetted and tested for arsenic by what might be termed the "mycotic method." Briefly this was done by placing in a 500 c.c. Erlenmeyer flask white bread (with crust removed) softened by distilled water,

upon which was scattered the carious debris. The flask was then sealed with a cotton plug and autoclaved for one hour. After cooling the bread was inoculated with the mold *Penicillium brevicaule,* and the flask was kept at body temperature for four or five days. At the end of this period, when opened, the flask contents gave a strong alliaceous (garlic-like) odor, which indicated that the mold had converted all or a major part of the arsenic present to volatile arsines. To ascertain whether the bread was negative, a control test was run on a similar sample, and this was entirely negative.

Inasmuch as corn kernels were known to contain arsenic, and as the carious material from teeth also showed its presence, the problem now was to produce caries by employment of correspondingly minute fractions of arsenic in the diet of albino rats. Therefore, in 1935 two attempts were made, using ten rats as variables and ten as controls; the basic diet used contained casein, starch, Mendel's normal salt mixture, cod-liver oil, and lettuce, giving freely distilled water in which had been dissolved 1 mg. of arsenic pentoxide per liter. Each group was fed for four months, and at autopsy all were negative. This result could be due to the great insolubility of arsenic in water, as well as the physical character of the diet.

Beginning Jan. 3, 1936, and ending March 15, 1936, a new approach was made, using eight rats as variables and eight as controls, and feeding a hard, flinty breakfast food (grape nuts) as the basic diet. The food for the variables was soaked for twenty-four hours in distilled water made alkaline with NaOH, and in which was dissolved sufficient arsenic trioxide to impregnate (theoretically) 1 kg. of the food with 1 mg. of arsenic trioxide. This wet mixture was then desiccated leaving a food of coarse, flinty consistency. The controls received the same diet minus the arsenic. Lettuce and carrots were fed biweekly, and rain water was given *ad libitum* to both groups. The inner leaves of lettuce only were fed to rule out possible arsenic contamination from insecticide spray. At the end of the feeding period one of the variables showed a huge cavity which had destroyed the whole crown of a lower molar. The controls were negative.

Beginning Sept. 30, 1936, and ending Jan. 21, 1937, nine rats were fed as mentioned in the preceding case. No controls were used. Six rats survived, three showing cavities. One of these cavities in a mandibular molar produced an area of disintegration of the lingual plate at the apices, evidently due to abscess formation with fistula. This area is about two millimeters in diameter, and a reproduction is shown in Fig. 1.

Beginning Feb. 1, 1937, and ending May 15, 1937, forty rats were fed as controls, the same diet being used as in the preceding case except, of course, no arsenic. Thirty-seven of these rats survived and showed no cavities.

Beginning Sept. 30, 1937, and ending Jan. 2, 1938, seventeen rats were used as variables. No controls were used. The regular basic diet plus 1 mg. of arsenic trioxide per kilogram of food was used. Fourteen of these rats survived, and of these five showed caries.

Beginning July 5, 1938, to Oct. 31, 1938, ten rats were used as variables and ten as controls. Six of the variables survived and at autopsy showed one large cavity and one small cavity. Seven of the controls survived and showed no cavities.

DISCUSSION

All of the rats used in this series were approximately six weeks old, and all were males. They were kept in a well ventilated and fairly well lighted room. The basic diet consisted of whole wheat, malted barley, yeast, and salt, and although not an ideal diet the majority of the rats thrived and gained weight.

It is admitted that the percentage of positive results is very small when compared with those of other investigators who fed corn meal. However, it must be remembered that they used a natural product, whereas the one used in these tests was synthetic. Furthermore corn kernels are tough and subject to better impaction, while this synthetic diet is friable and not subject to as effective impaction. Also, the arsenic content of corn is probably homogeneously distributed in the corn kernel.

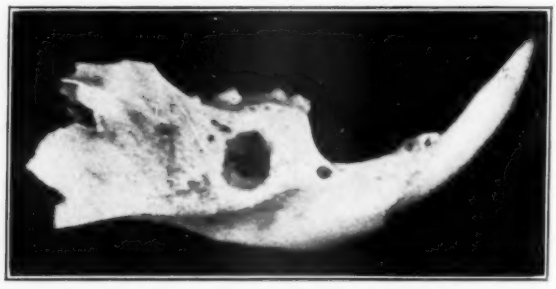

Fig. 1.

In conclusion it is realized that this series of investigations has not proved arsenic fractions as the specific causative agent in the production of dental caries, but it is believed that a new theory has been introduced, which may or may not prove tenable. Many will consider this theory fantastic—that trace quantities of this nature would be incapable of causing destruction of dental tissue. However, reflection will show that our knowledge of physical tolerance of such traces is not well developed. It should be remembered that carious lesions of the teeth are the result of an insidious destructive process, and therefore, it is well in the realm of possibility that a most minute particle of an element as destructively potent as arsenic might bring about this condition. This is a preliminary report, and the investigations are being continued.

I wish to extend my thanks to Professor Justin Powers of the School of Pharmacy, University of Michigan, in appreciation of his kindness in preparing the arsenic solutions used throughout the tests.

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104 S. W. Trick Building

UNERUPTED TEETH

STERLING V. MEAD, WASHINGTON, D. C.

CASE HISTORY

M. B., aged 12 years, was presented on Sept. 10, 1936, for full mouth roentgenograms, and a report was made to her dentist as follows:

The left maxillary third molar was impacted and extraction of this was indicated. The right maxillary second premolar was lingually displaced. Removal of this tooth was indicated. At this time exploration of the right maxillary supernumerary molar could be done, and the tooth removed. The right maxillary third molar was impacted, and extraction was indicated.

In the left mandibular second premolar region there was a supernumerary tooth. I recommended removing the second premolar and removing the supernumerary tooth at this time. There was an impacted mandibular third molar which will have room to erupt with the removal of the premolar. In the right mandibular second premolar region was a supernumerary tooth. Removal of this tooth and the molar was indicated to allow room for the eruption of the right mandibular third molor. The right mandibular second molar had a temporary filling. The left mandibular first molar had distal caries.

On Sept. 21, 1937, under an avertin anesthetic I removed the right maxillary second premolar and unerupted supernumerary premolar, the right maxillary impacted third molar, the right mandibular first molar, the right mandibular supernumerary tooth just anterior to the first molar, the left mandibular second premolar and supernumerary premolar, and the left maxillary impacted third molar. The postoperative x-rays taken immediately after this operation showed all of the areas to be clear, but there was a small area of condensation adjoining the roots of the right mandibular first premolar.

The patient was requested to return, and on July 12, 1938, a complete full mouth roentgenographic examination was done. In reviewing the case and looking over the roentgenograms, it was found that the original preoperative roentgenograms showed a slight area of rarefaction between the right mandibular first and second premolars near the gingival margin.

The roentgenograms taken on July 12, 1938 (a year after the patient was first seen) show that this tooth bud had developed, and on July 19, 1938, I removed this supernumerary tooth from the lingual surface as shown upon the postoperative x-rays.

1149 SIXTEENTH STREET, N. W.

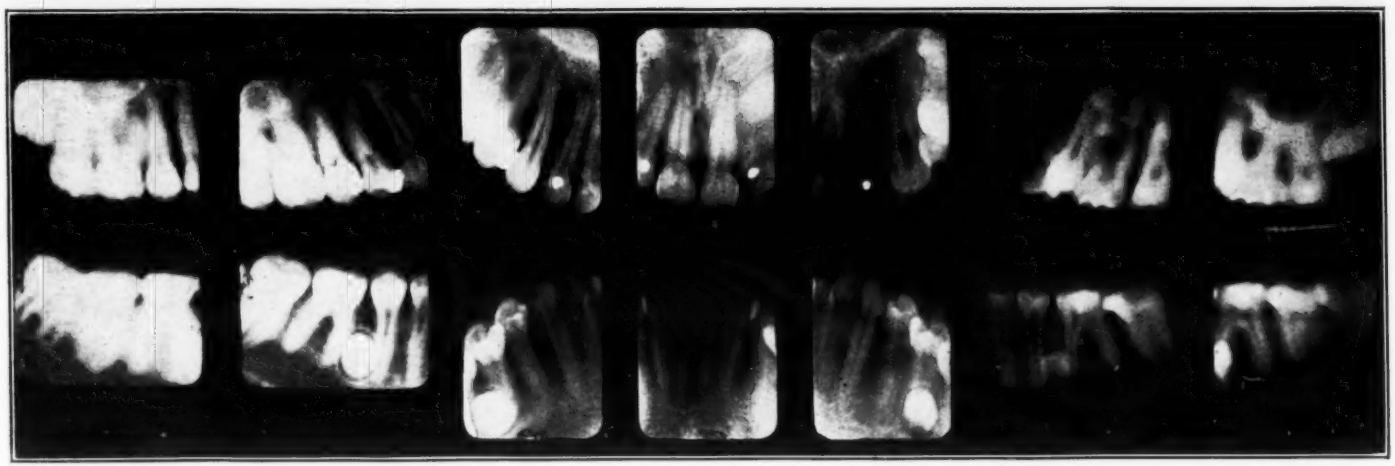

Fig. 1. (Above)—Complete mouth roentgenograms showing impacted teeth.

Fig. 2. (Right)—Intraoral roentgenograms taken Sept. 21, 1937, immediately after removal of impacted teeth.

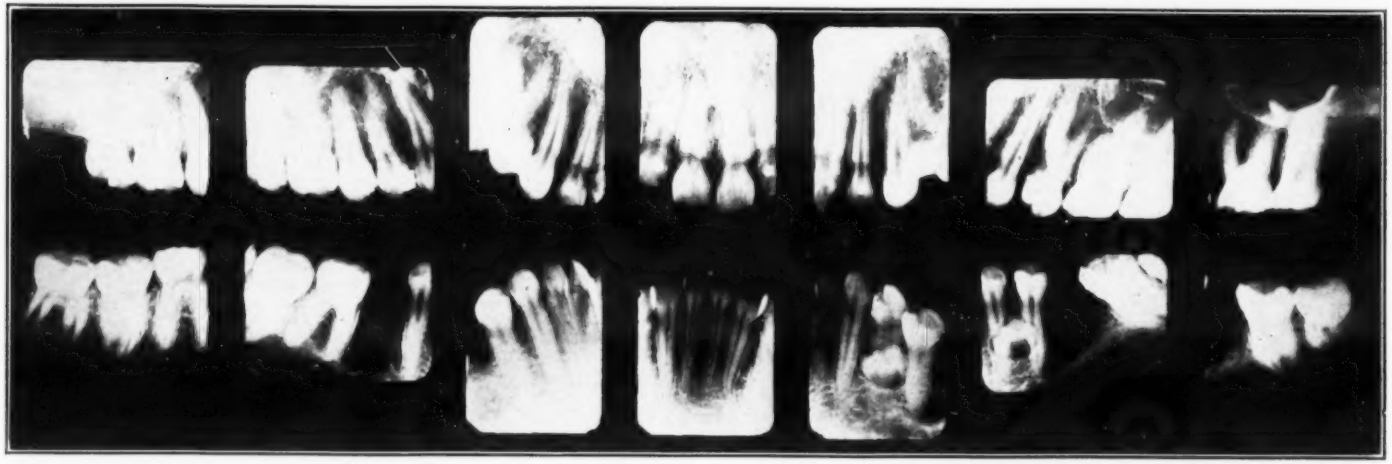

Fig. 3.—Complete mouth roentgenograms taken July 12, 1938, showing tooth bud which had developed in right mandibular premolar region.

Fig. 4.—Postoperative intraoral roentgenogram taken July 20, 1938, after removal of right mandibular premolar.

APLASTIC ANEMIA AND ALEUCEMIC LEUCEMIA

SIDNEY BRANDT,* D.D.S., NEW YORK, N. Y.

A MALE, an accountant, 28 years old, presented himself at Lincoln Hospital complaining chiefly of fainting and generalized weakness for a period of three weeks. Upon questioning the patient revealed that from the time his poor condition started, he noticed small blood clots in the interproximal spaces, and upon closer observation saw small streams of blood oozing from between his teeth. Thereupon the patient visited a dentist who applied adrenalin on ten different occasions, but in spite of this he sometimes awoke in the morning to find his pillow soaked with blood. The dentist believed that the patient was afflicted with some form of a Vincent's infection, and for this reason advised against extracting a maxillary incisor that had to be removed. However, upon the insistence of the patient, the tooth was removed two days prior to admission to the hospital. A great deal of hemorrhage resulted, and the patient returned to the dentist who packed the socket with but little success. The following day the patient vomited, then fainted, and upon the advice of a physician sought hospitalization.

Physical examination revealed no cardiac or pulmonary pathology. There was also no evidence of any vitamin deficiency. No family history of hemophilia, cancer, or tuberculosis was determined. Radiographs of the teeth revealed three devitalized teeth and a retained root, but no evidence of bone pathology. A mouth smear was negative to Vincent's organisms. The important features of the blood picture were as follows:

Red blood cells, 1,900,000; white blood cells, 1,200; platelets, 10,000; lymphocyte percentage, 69 per cent. (This is obviously a marked rise over a usual normal per cent of 20-30.)

Upon the clinical observations and blood picture, the diagnosis of aplastic anemia and aleucemic leucemia was made. The prognosis in these diseases is usually fatal.

This case should be of extreme interest to the dentist as it is a classic example of how he can render great service to patients. Careful history taking will reveal much where the operator is suspicious of anything systemic. Closer cooperation with the medical profession will help patients and will strengthen our own profession.

*Dental Interne, Lincoln Hospital.

WELLS OVER MORTON—OR A CRITICISM OF THE BOOK "TRIUMPH OVER PAIN"

WILSON R. CONRAN, D.D.S., HARTFORD, CONN.

I HAVE read with interest and with profit the very excellent book "Triumph Over Pain," in which the author dramatizes with great effect the long struggle to produce an anesthetic suitable to medical and dental surgery. In its broad lines the book tells so accurate a story that I do not like to quarrel with any of its facts, but I should like to take exception to one item at least, namely, the giving of credit to Dr. Morton for the discovery of the possibility of the use of an anesthetic. Indeed, I believe that the author is wrong in his emphasis, if not in his facts in telling the entire story of the interesting history of the introduction of anesthesia.

The work pays great honor to various individuals who worked on chemicals to produce nitrous oxide, but I feel that some recognition should be given to the courageous and scientific spirit of Sir Humphry Davy of Bristol, England, who tried out the gas on himself when it was commonly assumed that inhaling it meant certain death. This was in 1799; in 1800 he inhaled nitrous oxide when he was cutting a wisdom tooth and, while under the influence of the gas, experienced no pain. He wrote a book on his experience, stating that the use of nitrous oxide was in its infancy; apparently he did not realize what he had accomplished nor how nitrous oxide could be applied to relieve pain in surgical operations.

Professor G. Q. Colton gave a course of lectures in chemistry and philosophy in Hartford in 1844 and amused his audience with nitrous oxide. He administered gas to anyone who presented himself as a subject. Dr. Horace Wells was a subject, much to his wife's annoyance. Besides watching its effects on others, he wished to have personal experience with the gas. A very prominent Hartford man was a subject, and he fell over chairs and benches barking his shins while under the influence of the gas. Upon coming out of the effects of the gas, he had no recollection of his doings and experienced no pain in his legs, though they were found on examination to be bleeding as a result of his antics. This performance suggested to Dr. Wells the possibility that tooth extractions or limb amputations might be performed under nitrous oxide anesthesia. Dr. Wells asked Professor Colton if a tooth might be extracted under nitrous oxide anesthesia, and the professor admitted that, although he had been giving demonstrations for over a year, that thought had never occurred to him. The following day in Dr. Wells's office in the Old Union Hall building, 162½ Main Street, Hartford, Conn., Dr. J. M. Riggs extracted a maxillary third molar while Dr. Wells was inhaling nitrous oxide.

Dr. E. E. Marcy of Amherst College, also spoken of by the author as a discoverer of gas and ether, did not know about the use of the gases until

he was shown by Dr. Wells how he extracted teeth with gas. He then suggested to Dr. Wells that ether could be prepared much easier. They removed a tumor from a patient's head, but, like Professor Rolger of Washington (now Trinity) College, both thought ether was more dangerous than nitrous oxide.

Then in January, 1845, Wells went to Boston to make known his discovery. There he addressed the Medical College and gave gas to a boy, removing a tooth. The boy had a prolonged excitement period, and as a result, the cry of fraud was called. "Triumph Over Pain" failed to state that the boy admitted he knew absolutely nothing about the extraction, but because he cried out, they put the demonstration down as a failure. The author of "Triumph Over Pain" must be aware that even in the present day a patient will cry out and carry on during the excitement period, making a lay person feel the patient is not under the influence of gas.

Now, as to the great Dr. Morton, he was nothing more than a pupil of Dr. Wells in dentistry in 1841 and 1842 and during that time resided in Farmington, Conn. He then became associated with Dr. Wells until Oct. 18, 1844, when the association was dissolved. Morton went to Boston entering the office of Dr. C. F. Jackson. Dr. Jackson was then noted as a chemist and was of the same opinion as this medical student, that extractions of teeth and surgery were not to be accomplished with nitrous oxide. Morton was present when Dr. Wells gave the demonstration in Boston. He then came to Hartford and talked with Wells and Riggs about gas and wanted them to give him some and to show him how it was prepared. Dr. Wells referred him to Dr. Jackson, who could prepare and show him how.

In 1846 a Miss Elizabeth Williams of Hartford met Dr. Morton in Stafford Springs, Conn., and hearing he was a dentist, told him about the extraction of a tooth by Dr. Wells the previous year. He asked her about the effect and how the gas was administered and gave no intimation that he had any knowledge of any anesthetic.

In September, 1846, the Boston papers came out with an article giving Morton and Jackson credit for discovering an anesthetic under which a Mr. Eben Frost had a tooth extracted. Dr. Wells at once made known to Dr. Jackson the fact that he and Dr. Marcy used ether two years previously.

Drs. Morton and Jackson then tried patenting the ether formula in October, 1846, but before the patent could be granted, Jackson withdrew his name, fearing he might be censored by the medical society. However, he bound Morton to pay him 10 per cent of all the profits; he later wanted 25 per cent. The patent was granted in November, 1846. They had circulars printed proclaiming them the discoverers and inventors of sulphuric ether. Agents were sent out and this character, to whom the moving picture people and many in and about Boston would give credit for a discovery, was pushed forward by the press, over the rights of his old teacher, Dr. Wells.

The Paris Medical Society at first gave Morton credit but later, on evidence presented, awarded Dr. Wells a diploma and the vote of the Society.

Both Jackson and Morton, from the start, claimed nitrous oxide was no anesthetic and was a failure. They ignored the fact that Dr. Wells and Dr. Marcy had used ether but discontinued its use as they felt nitrous oxide was safer and easier to take.

Morton tried to have a bill pushed through Congress naming him the benefactor of the Army and Navy and bestowing honor on him for the use of ether, but this was defeated by the efforts of our Connecticut Senator, the Honorable Truman Smith.

In 1847 the Connecticut General Assembly passed a resolution in favor of Dr. Wells as the discoverer of anesthesia and declared him entitled to the favorable consideration of his fellow citizens. The Court of Common Council of Hartford passed a resolution to the same effect. So this man was considered great even in his own backyard, thus showing the old adage, "A prophet is without honor in his own country," is not always true.

Toward the last, Dr. Wells, due to worry and annoyances by different anesthesia agents, had a series of mental disturbances, and under this influence, put an end to his suffering Jan. 24, 1848. I believe the handling of this great man's end by the author of "Triumph Over Pain" could have been much more tactful, taking into consideration his mental state at this time and his many sufferings.

From this brief paper, any one can see the difference in the two men in question and what each really accomplished. Wells had the courage of his convictions in that he was willing to have a tooth extracted under this new anesthetic which so many thought was dangerous and not even to be classed as an anesthetic. Morton, on the other hand, was willing to take the credit and any financial reward even if it would mean stepping over an old friend and teacher. There is no doubt that any fair-minded person would unquestionably place the honor of being the father of anesthesia on Dr. Horace Wells.

750 Main Street

Case Reports

TRAUMATIC (AMPUTATION) NEUROMA

LESTER R. CAHN,* NEW YORK, N. Y.

THIS case is reported because I have seen nothing in the literature concerning the formation of neuromas about traumatized maxillary or mandibular nerves, with the exception of the case recounted by Dr. Thoma in his book "Clinical Pathology of the Jaws" (page 366).

Several years ago I removed from the region of the left mental foramen what I thought was scar tissue engulfing nerve bundles. The patient was an edentulous, middle-aged spinster, who had suffered from reflex neuralgia that had radiated from the involved area. The pain had spread along the mandible, up through the side of the face, and into the eye on the affected side. There also had been pain in the throat which had been diagnosed as chronic pharyngitis, and for which she had received treatment.

Clinically, there were in the mental region small, painful, shotlike masses. On their removal the reflex neuralgia disappeared. Histologic examination showed the masses to be composed of dense fibrous connective tissue surrounding rather large nerve bundles (Fig. 1).

About one year later the patient returned, complaining of her old trouble. The region of the mental foramen was infiltrated with 2 per cent novocain, without suprarenin—a procedure which brought considerable relief.

All went well for another year, when the condition returned. This time it was a little severer than before, and the pain in the eye was especially marked. This gave her great concern, since she had had an acute glaucoma in this eye a number of years previously, for which she had been successfully operated upon. However, her ophthalmologist could find no evidence of recurrence.

On examination of the mental region a movable, submucous, painful nodule, about the size of a green pea, could be palpated. Pressure on this small mass caused pain along the distribution of the inferior dental nerve and also in the ear and maxilla.

Under mandibular block and infiltration anesthesia, with a 2 per cent novocain suprarenin solution, an incision was made over the crest of the alveolus and so curved as to encircle the mental foramen and the growth. With blunt dissection the mucoperiosteum was pushed down until the mental foramen and nerve

*Chairman of the Division of Oral Pathology, Columbia University.

were exposed. At this point, since pain was felt when the exposed nerve was touched, novocain was injected directly into the nerve, which was seen to emerge from the foramen and enter a bulbous mass. The nerve was grasped with a hemostat, somewhat evulsed from the foramen, and then cut. The severed portion of the nerve, together with its bulbous end, was dissected from the surrounding soft tissue. The structures in the foramen were destroyed with the actual cautery. The wound was closed with one stitch, and a small drain was inserted.

Fig. 1.—The tissue removed at the first operation. *A*, Large nerve trunk, *B*, dense fibrous connective tissue.

There was a variable amount of pain after the operation, but by the fifth postoperative day she was entirely free from all reflex neuralgia, and her eye, which had given her her greatest worry, no longer bothered her. Numbness in the lip was apparent.

About fifteen days later the wound had entirely healed, and she was able to wear her lower denture comfortably. Paresthesia, determined by a pin prick, was present in the lip and in the mucous membrane of the mandible in an area extending from about the left first premolar to the lateral incisor on the buccal and occlusal surfaces. There was naturally no lingual anesthesia. She is at present extremely comfortable and does not mind the numbness in the lip.

PATHOLOGIC EXAMINATION

On cutting the removed specimen in half, one could see the severed portion of the mental nerve entering a small fibrous-looking mass which was surrounded by a tissue of different texture.

Histologic examination disclosed that the small growth was composed of nerve bundles separated by fibrous septa and growing in a more or less disorderly fashion. These were a continuation of the axons and their sheaths of the mental nerve and represented a hyperplasia and a hypertrophy of these structures. The mass was embedded in fibrous connective tissue (Figs. 2 and 3). The diagnosis was traumatic (amputation) neuroma.

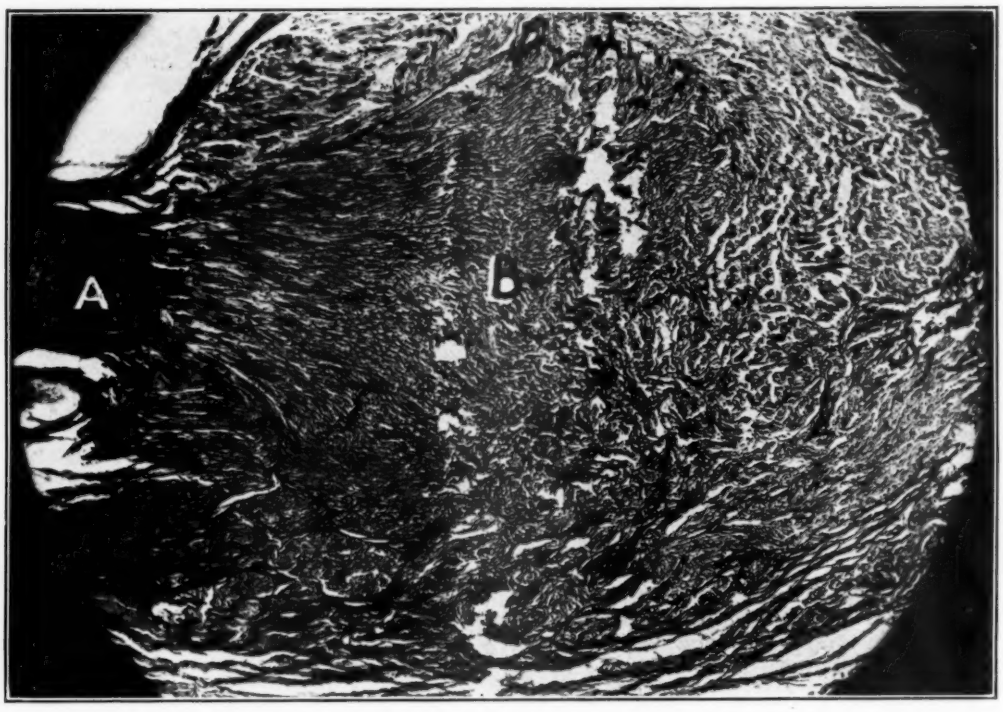

Fig. 2.—Low power view showing the relationship between the mental nerve and the amputation neuroma. *A*, Mental nerve, *B*, amputation neuroma.

COMMENT

The amputation neuroma is not a true neoplasm and is sometimes spoken of as a false neuroma. It is the result of an attempt at repair of a severed nerve. When a nerve is cut, the myelin sheaths of the proximal end degenerate only for a short distance—supposedly only to the next node of Ranvier. Then the connective tissue cells of the perineurium and endoneurium, as well as the Schwann cells and axons, proliferate in an attempt to connect with the distal end of the cut nerve. When they cannot, they form a bulbous mass of tangled interlacing fibers, which becomes embedded in connective tissue.

It is amazing that more lesions, such as these, have not been reported, since they must exist in the jaws of many patients. I have examined, histologically, a number of cyst membranes removed from the regions of the mental and anterior palatine foramina and have found severed nerve bundles in these "sacs." It is not unreasonable to suppose that traumatic neuromas may have developed on the proximal ends of the cut nerves. I know of one instance where this actually happened after the removal of a cyst near the mental foramen.

A particularly difficult or vicious extraction in the neighborhood of an emerging nerve, also, may well result in a traumatic neuroma, and it is probable that much reflex neuralgia may be caused by the presence of unsuspected neuromas.

In seeking a cause for reflex pain, one should palpate carefully in the region of the orifices of emerging nerves to note whether a neuroma is present.

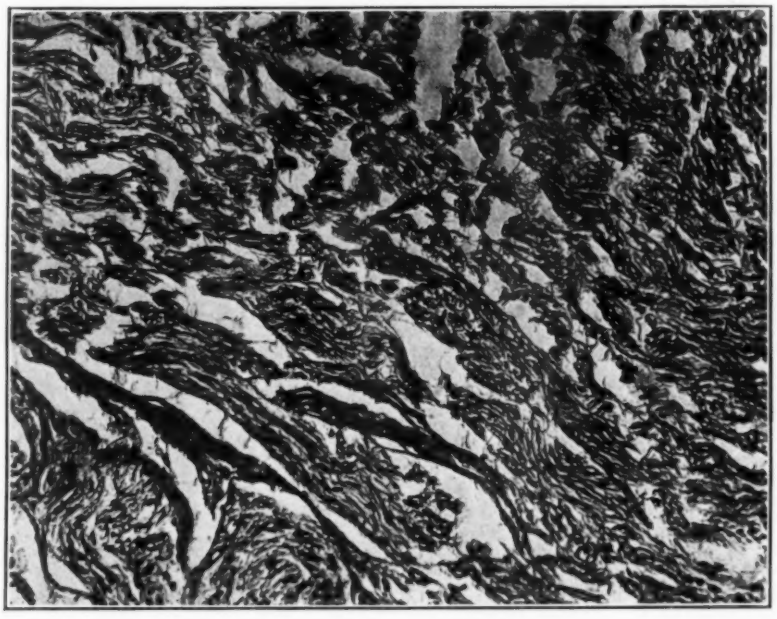

Fig. 3.—Higher power view of the neuroma. Note the irregular arrangement of the nerve fibers, embedded in fibrous connective tissue.

As far as the removal of the lesion is concerned, the technique described is satisfactory, but nerves have a great tendency to regenerate and proliferate, and the greatest problem is to prevent the recurrence of another neuroma. I attempted to do this by destroying the contents of the mental foramen with the cautery, which I hope will be successful. A better method, and one that I remembered only after the operation had been completed, would have been to put a sterile plug of wood in the foramen and thus block it up. This is the procedure that neurosurgeons use when they resect the various divisions of the fifth nerve for the relief of trigeminal neuralgia.

SUMMARY

A case is presented of an amputation neuroma of the mental nerve, that had caused reflex neuralgia. The pathology of the neuroma is discussed. The possibility of unsuspected neuromas following dental surgical procedures, which may be the cause of obscure pain, is considered. A technique is suggested for preventing the recurrence of a neuroma by blocking the foramen with a sterile plug of inert material.

944 Park Avenue

Department of Orthodontic Abstracts and Reviews

Edited by

Dr. Egon Neustadt, New York City

All communications concerning further information about abstracted material and the acceptance of articles or books for consideration in this department should be addressed to Dr. Egon Neustadt, 133 East Fifty-Eighth Street, New York City.

An Histologic Study of the Investing Tissues of the Teeth, the Dentine, and Dental Pulp. By Professor H. H. Stones, M.D., M.D.S.Manc.,L.D.S. Eng. (From the Research Department, School of Dental Surgery, Liverpool University). Abstract of original article published in The British Dental Journal **65**: 711-744, December, 1938.

For the purposes of this investigation traumatic occlusion was produced by inserted raised fillings in three posterior teeth of six monkeys. In a seventh monkey a raised crown was fixed on an upper central incisor. Experiments were terminated at varying periods of time from ten to forty-three weeks, after which sections were prepared for histologic examination.

The changes in the periodontal membrane, alveolus, cementum, dentine, and pulp are described.

Depending on the direction of the force, the periodontal membrane may be either compressed or stretched (Fig 1). The tissues usually endeavor to regain the physiologic width of the periodontal membrane by resorption of the alveolus on the compressed side and deposition of new bone on the other (Fig. 2).

The reaction of the alveolus to increased stress may be either resorption or sclerosis (Fig. 2). Resorption would appear to be usually associated with the pathologic changes of parodontal disease in the subgingival epithelium, while with sclerosis, the gingival tissues tend to remain normal.

If the alveolus between two roots is completely resorbed so that the latter are nearly adjoining, one of two reactions may occur: A. Provided there is still an intervening strip of the remains of the periodontal membrane, the cementum and then the dentine of the root are resorbed (Fig. 1). When the stress is equalized, new cementum is deposited on the denuded end. B. If the intervening strip of the periodontal membrane is destroyed, there is no further resorption.

The changes in the cementum, except when the roots are in contact, as a result of the intervening alveolus having been resorbed, are only slight.

Additional growth of primary dentine under the occlusal surface of premolars and molars is produced as a result of the increased stress in teeth oppos-

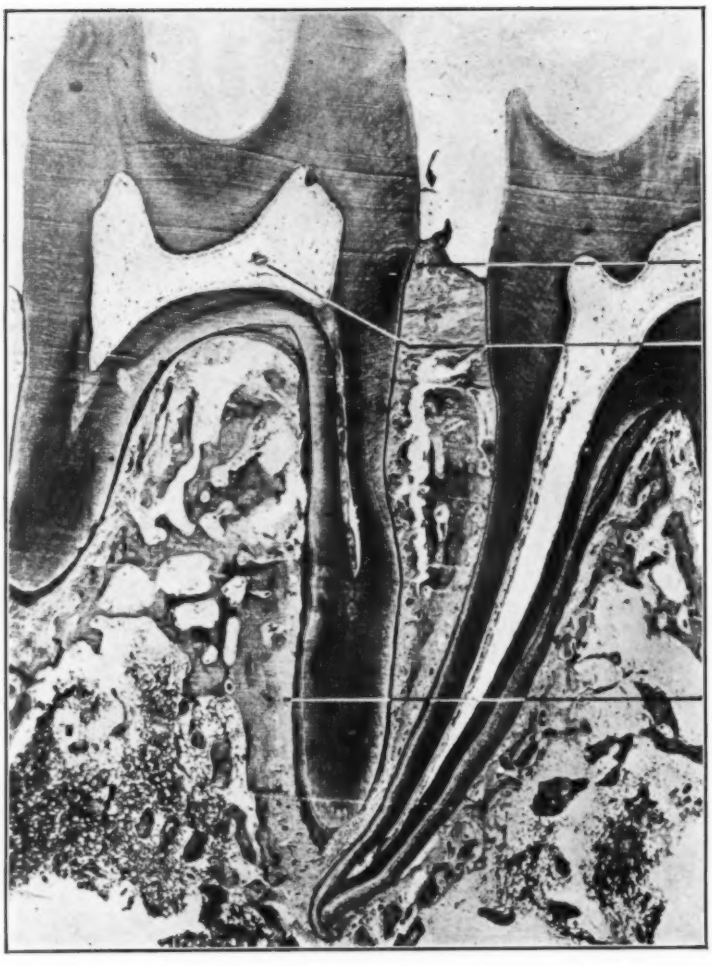

Fig. 1.—$\overline{|\,67}$ opposing raised fillings in $\underline{|\,567.}$ General view. Extreme stress on $\overline{|\,6}$ has forced the root against $\overline{|\,7}$ and partly resorbed the apex. The periodontal membrane *P.M.* is stretched on the opposite side. There is proliferation of epithelium *E.* along the cementum of $\overline{|\,6.}$ A hemorrhage *H.* is seen in the pulp. ×10.

ing those with raised fillings. The teeth in which cavities were prepared show much more drastic changes, including various kinds of secondary dentine (Fig. 2).

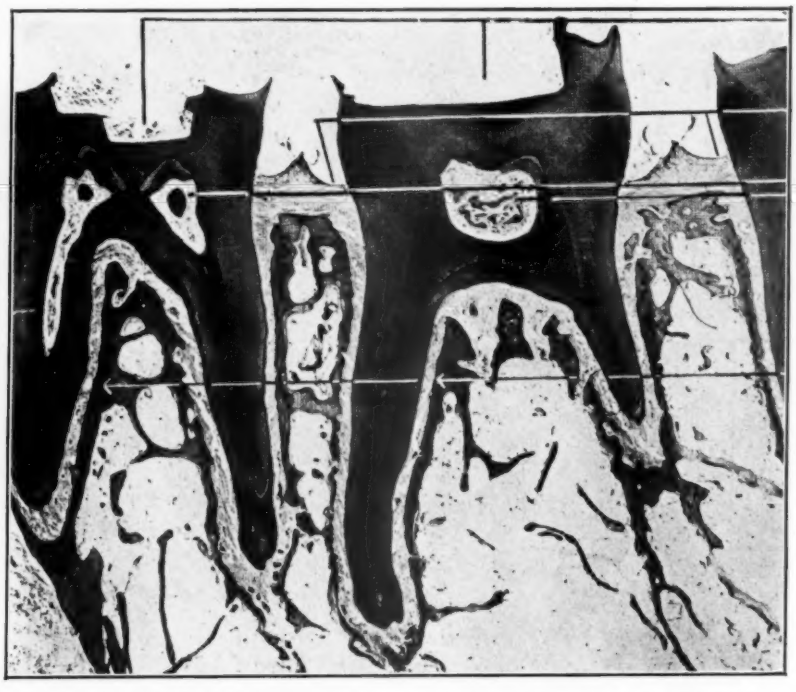

Fig. 2.—Raised fillings in 6|, 7|, and 8|. Greatest stress on 7|. Duration twenty-eight weeks. The cavities *Cav.* are seen. 7| is the only tooth that shows pathologic changes in the subgingival epithelium *E.* In 6| and 7| the alveolus *A.* is irregular, and in 6| it is sclerosed. The periodontal membrane has regained its physiologic width. All pulps contain secondary dentine *S.D.*, and 6| also shows two pulp stones *P.S.* ×10.

In the pulp increased stress alone tends to produce hemorrhages (Fig. 1) and pulp stones (Fig. 2). These hemorrhages would appear to be associated with the formation of the latter.

Degeneration and death of the odontoblasts and cells of the pulp are recorded, also fibroid and possibly lipoid degeneration and cystic formation.

Hodgen-Shell Dental Materials. By John S. Shell, B.S., M.A. Published by The C. V. Mosby Company, St. Louis, 1938.

As dental procedures become more exacting, likewise the materials used by the dentist must meet more exacting demands. Dental materials, especially the metals and their alloys, are becoming more numerous and complex. All materials concerned with the restorative phase of dentistry are discussed and evaluated by the author.

Dental materials can be divided into three general groups: (1) metals and alloys, (2) inorganic compounds; such as plaster of Paris, silicates, ceramic compounds, etc., and (3) organic compounds; waxes, resin compounds, gutta percha, etc.

More pure metals, the author tells us, are used in dentistry than in any other art or science, because of the special requirements that present themselves in dental practice. Alloys, especially those combined with gold, are continually undergoing modifications by the various manufacturers, so as to meet new demands and to improve their characteristics. The reputable manufacturers of dental golds are continually improving their methods so that the dentist may be assured of a better product.

When the "steels" are used for orthodontic purposes, it is necessary that they contain a low chromium content so that they may respond to heat treatment. Unfortunately this causes the steel to be unresistant to the action of saliva. Soldering stainless steel causes discoloration and disintegration. The only reliable method of attaching steel is by spot welding. The author admits that in orthodontic practice the use of steels is more promising than for any other branch of dentistry.

Mr. Shell as director of research of a large gold manufacturing firm has been able to observe the practical effects of the theoretical observations he has made. The student and dentist will be rewarded with a clearer conception of the theory of metals and materials by a careful study of this excellent work.

S. Trieff.

Editorial

Lay Dental Education With Cooperation of the Greater New York Bureau for Dental Information

IN PUBLISHING the interesting pictorial story of modern dentistry and in making the assertions that since 1920 the science of dentistry has advanced at a tremendous pace and that the United States outclasses all other nations in the quality of its dental care and in featuring the fact that, on account of the continuous trend of civilization toward a more artificial trend of life, the teeth of human beings have become a major health problem, *Life* magazine in its issue of Jan. 16, 1939, devoted ten pages to what will, no doubt, prove to be the most outstanding, constructive, and educational story for the layman that has ever been published in any publication of wide lay distribution.

Life's approach is different in that it vividly portrays the wide purpose and field of modern scientific dentistry and also dramatizes the subject for reader interest. Unfortunately, dentistry has never enjoyed the financial backing of large foundations and benefactions as have many other departments of science; notwithstanding this, it has been fortunate in recruiting among its workers enthusiasts with a purpose and loyalty to its ideals that have been surpassed by few activities that pertain to the physical well-being of mankind.

It is not so well understood that many of the foremost advances in dental progress have been worked out, step by step, within the four walls and unpretentious setting of someone's private dental office, many times at private expense, and the whole benefaction then given to the profession in the spirit of "for the good of the order and for the good of humanity." It is not so well known, for instance, that many of the important advances made by dentistry have been accomplished in dental schools, entirely dependent upon their own financial resources, and many times the research work has been done by part-time teachers whose revenue is derived almost entirely from their private practices.

It is recalled that in his presidential address before the American Dental Association in San Francisco, July 14, 1936, Dr. George B. Winter made the assertion that the American Dental Association desires to give the public only the facts pertaining to dental topics and to do so in such a way that the facts can be perfectly understood. He pointed out that it is cruel for the expectations of the public to be aroused by fanfare publicity to the point that it expects more than can be delivered but that the time has now arrived when the public should know by means of carefully directed publicity just what has been accomplished and what is to be expected of modern, scientific dentistry.

The merit of the above theme has been amply proved in worth-while news interest as demonstrated in *Life* magazine. It is interesting to know that the whole gesture required three months of preparation, and that the story was worked out by the Greater New York Bureau for Dental Information in conjunction with the editors and photographers of *Life* magazine. The Bureau deserves much credit because it arranged for the facilities for taking photo-

graphs, checked, and also supplied the magazine with many facts and material. Dr. C. Franklin MacDonald, chairman of the Bureau, and Dr. Edwin G. Van Valey, representing the New York Academy of Dentistry, and Mr. Fice Mork, public relations counsel of the Bureau, did much of this work and have thereby helped to paint the modern picture of dentistry on public consciousness as it should be.

It is interesting as well to know that 300 photographs were taken during the period of preparation to illustrate this story. Much credit is due to science editor Andrew Heiskell, photographer Otto Hagel, and researcher Will Lang of *Life's* staff for the sympathetic, genial, and unhesitating cooperation with which they worked with the Greater New York Bureau for Dental Information during the preparation of this important essay on dentistry. This is a constructive piece of work for the public and for dental science.

H. C. P.

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Harvard Medical School
Shattuck St., Boston, Mass.
December 19, 1938

H. C. Pollock, D.D.S., Editor
American Journal of Orthodontics and Oral Surgery
The C. V. Mosby Co.
St. Louis, Mo.

Dear Dr. Pollock:

In the AMERICAN JOURNAL OF ORTHODONTICS AND ORAL SURGERY, Vol. 24, No. 4, pp. 328-336, April, 1938, Dr. Hirosi Iwagaki in his paper entitled "Hereditary Influence of Malocclusion" states:

"Dr Keeler is probably the first man who studied those problems from the point of recent advances of the genetic sciences, *but without any decisive conclusion.*" [Italics mine.]

I am a bit puzzled by Dr. Iwagaki's interpretation of my paper because *I stated very definite conclusions.*

On page 1161 in my monographic article in *Dental Cosmos,* referred to by Dr. Iwagaki, I stated:

"*Fifteen of them give abundant and undoubted evidence of heredity.*"

And on page 1162 I said:

"There is undoubtedly one and possibly a second sex-linked character. *The rest are unit dominants.* The majority are orthodox dominants. Some produce a few normal overlaps. One pedigree of harelip suggests a recessive unit-character in contrast to its dominant nature in three other pedigrees."

Because Dr. Iwagaki's statement published in your journal will have wide circulation and will go far to negate the value of my 1935 paper, which was the first to point out that many dental variations are definitely hereditary and that they do obey the simple laws of genetic transmission, I should like to request that you print this letter in an early number of the AMERICAN JOURNAL OF ORTHODONTICS AND ORAL SURGERY.

Very truly yours,

(Signed) *Clyde E. Keeler.*

News and Notes

American Association of Orthodontists

The Thirty-Seventh Annual Meeting of the American Association of Orthodontists will be held in Kansas City, Mo., April 17-20, 1939.

CLAUDE R. WOOD, Sec.-Treas.
Medical Arts Building
Knoxville, Tenn.

The American Board of Orthodontia

A meeting of the American Board of Orthodontia will be held in Kansas City, Mo., Saturday, April 15, 1939. Orthodontists who desire to qualify for certificates from the Board should secure the necessary application blanks from the secretary. The applications must be returned to the secretary, together with any other required credentials, prior to the date of examination. Applications filed at the time of the Board meeting will have preliminary consideration, so that the applicant may be advised of the work required for his subsequent examination.

Attention is called to the following resolutions adopted by the Board: Any person desiring to make application to the Board for a certificate must have been in the exclusive practice of orthodontics for a period of not less than five years or an equivalent to be determined by the Board and based upon the following conditions:

1. He must be an instructor in orthodontics in a school satisfactory to the Board.
2. He must be an associate in the office of an orthodontist whose standing is satisfactory to the Board.
3. It is to be definitely understood that any person at the time of making application for a certificate shall be in the exclusive practice of orthodontics in his own name.

For further information please address:

CHARLES R. BAKER, Secretary
636 Church Street
Evanston, Ill.

Orthodontists Certified

Certificates of qualification were granted to the following orthodontists by The American Board of Orthodontia in 1938:

Dr. Culmer Coleman Benton, San Diego, Calif.
Dr. Walter Coolidge Chapin, New York, N. Y.
Dr. William T. Chapman, El Paso, Texas.
Dr. George Campbell Chuck, Long Beach, Calif.
Dr. Edwin Grant Flint, Pittsburgh, Pa.
Dr. William Revis Flint, Pittsburgh, Pa.
Dr. C. S. Foster, Cedar Rapids, Iowa.
Dr. Emery James Fraser, Seattle, Wash.
Dr. Guy Melton Gillespie, Abilene, Texas.
Dr. I. Eugene Gould, Pasadena, Calif.
Dr. Alfred R. Higson, Beverly Hills, Calif.
Dr. Sidney Blaine Hoskin, Portland, Ore.
Dr. Walter Lloyd Hyde, Minneapolis, Minn.
Dr. Edmund M. Johnston, Riverside, Calif.
Dr. Jesse A. Linn, Los Angeles, Calif.
Dr. Fred E. McIntosh, Los Angeles, Calif.
Dr. Philip T. Meaney, Portland, Ore.
Dr. Hays N. Nance, Los Angeles, Calif.
Dr. Francis William Nash, Scranton, Pa.
*Dr. Ray W. Noland, Des Moines, Iowa.
Dr. Harold Edward Sippel, Buffalo, N. Y.
Dr. Harvey Stallard, San Diego, Calif.
Dr. Charles H. Tweed, Jr., Tucson, Ariz.
Dr. James Thompson Walls, Portland, Ore.
Dr. E. Forris Woodring, Tulsa, Okla.

HARRY E. KELSEY, President
CHARLES R. BAKER, Secretary

*Posthumous.

University of Pennsylvania Postgraduate Courses

The School of Dentistry of the University of Pennsylvania offers two special postgraduate courses as follows:

1. Four-day general refresher course, including local and conduction anesthesia, radiography, bacteriology, full-denture prosthesis, partial-denture prosthesis, ceramics, gold inlay, periodontia. The course will be conducted by the faculty of the school, aided by well-known specialists in the fields concerned, from Monday, June 5, to Thursday, June 8, 1939, inclusive. The fee is $10, payable with application.

2. Ten-day course in oral surgery, exodontia, and local and general anesthesia. The course will consist in part of lectures on the subjects mentioned, supplemented by lectures and demonstrations in anatomy, bacteriology, pathology, and roentgenology, pertinent to the clinical subjects. Most of the time will be spent in clinical instruction at the Evans Institute and at various hospitals. This course will be held Monday, May, 29, to Thursday, June 8, 1939, inclusive. The fee is $75, $25 to be sent with application and $50 payable at time of registration at the school.

Applicants should state their school and class and dental society affiliation. Courses are not necessarily restricted to University of Pennsylvania graduates, but alumni will be given preference in case the numerical limits are exceeded.

Application for either course should be made to

THE DEAN, School of Dentistry, University
of Pennsylvania
Evans Institute, 40th and Spruce Streets
Philadelphia, Pa.

French Speaking Dentists of North America

The fifth convention of the French Speaking Dentists of North America will be held at Montreal, Mount Royal Hotel, June 8-10, 1939.

DR. GERARD BAILLARGEON
Medical Arts Building
Montreal, Canada

Cleveland Dental Society

The Annual Spring Clinic Meeting of the Cleveland Dental Society will be held April 3 and 4, 1939, at Hotel Statler.

LESTER G. GLICK
562 Rose Building,
Cleveland, Ohio

Annual M. I. Schamberg Meeting

The third annual M. I. Schamberg Meeting, under the auspices of the Bronx Hospital Dental Staff, will be held at 8:30 P.M. Thursday, April 6, 1939, at the Bronx Hospital, Fulton Avenue at 169th Street, Borough of the Bronx, New York City. Dr. Robert H. Ivy of Philadelphia will speak on "Pathological Conditions of the Face, Mouth, and Jaws" illustrated by lantern slides in color. Dr. Harold S. Vaughan of New York and Dr. M. I. Schamberg of New York will discuss the paper. All members of the dental profession are invited to attend.

S. M. FRANK, Secretary,
Committee of Arrangements.

Massachusetts Dental Society

The seventy-fifth annual meeting of the Massachusetts Dental Society will be held at the Hotel Statler in Boston, Mass., April 24-27, 1939. The subject of the meeting will be the "Trend of Dentistry During the Last Seventy-five Years." There will be outstanding speakers from all over the country, a health exhibit, and a clinical program.

DR. FRANK W. ROUNDS, President,
270 Commonwealth Avenue,
Boston, Mass.

Chicago Dental Society

The 1939 Midwinter Meeting of the Chicago Dental Society will be held February 13-16 inclusive at the Stevens Hotel. The meeting will once again return to its four-day length after an experiment with a five-day session last year. In 1938 the Midwinter Meeting achieved new records in attendance, with more than ten thousand visitors being registered during the five days. Greatly enlarged commercial and scientific exhibits were also features which will be continued for the 1939 meeting.

Central Society of Orthodontists

The Central Society of Orthodontists will have a luncheon meeting at the Stevens Hotel in Chicago, Monday noon, Feb. 13, 1939.

L. B. HIGLEY
Secretary-Treasurer

Southwest Dental Congress

At the Southwest Dental Congress, the eight states postgraduate meeting, will offer 180 hours postgraduate study in nine sections in Oklahoma City, Okla., April 24-28, 1939.

HARRY H. SORRELS
General Chairman
Medical Arts Building
Oklahoma City, Okla.

Southwest Dental Assistants Association

The Southwest Dental Assistants Association will meet in Oklahoma City, Okla., April 24-28, 1939.

Pacific Coast Society of Orthodontists

The biennial meeting of the Pacific Coast Society of Orthodontists will be held at the Palace Hotel, San Francisco, Calif., Feb. 21, 22, and 23, 1939. Practitioners of orthodontics exclusively are cordially invited.

DR. EARL F. LUSSIER, Secretary-Treasurer
450 Sutter Bldg.
San Francisco, Calif.

Southern Society of Orthodontists

The seventeenth annual meeting of the Southern Society of Orthodontists will be held at Hotel De Soto, Savannah, Ga., on March 6 and 7, 1939. All ethical members of the dental and medical professions are cordially invited to attend.

Dr. Sam G. Cole, President
Medical Arts Building
Atlanta, Ga.

Five State Post Graduate Clinic

The Annual Five State Post Graduate Clinic will be held March 5-9, 1939, at the Mayflower Hotel, Washington, D. C. Exhibits, clinics, features, and frolic will be included in the program.

A. G. Miller, Chairman
Publicity Committee